The Journal of Thoracic Surgery

VOL. 6 DECEMBER, 1936 NO. 2

Original Communications

NONTUBERCULOUS ABSCESS OF THE LUNG

ETIOLOGY, TREATMENT, AND RESULTS IN 90 CASES*

ELLIOTT C. CUTLER, M.D., AND ROBERT E. GROSS, M.D.
BOSTON, MASS.

NONTUBERCULOUS pulmonary abscess must seem a dry and prosaic subject to bring before this Association whose existence has been simultaneous with the period of greatest development of intrathoracic surgery. The many brilliant papers that have appeared here in the fields of pulmonary and cardiac surgery, culminating in pneumectomy for tumor and several new methods for a direct attack upon the heart and pericardium, make pulmonary abscess a seemingly humdrum topic. But we must not lose sight of the fact that it is a common disease and one still carrying a high mortality. Moreover, it is a disorder handled largely by the general surgeon who looks to us in this Association for guidance in the improvement of therapeutic measures. That we have not been unmindful of our responsibility in this field is shown by the fact that many studies dealing with both the therapy and the pathogenesis of abscess of the lung have frequently appeared in our transactions.

It is true that our personal interest has largely centered upon the etiology of abscess of the lung, since only by an understanding of how it arises can we prevent the occurrence of this condition. At the same time, there have been contributions to our therapeutic methods which make us feel that it is worth while to restudy a fairly large and carefully controlled group. It appears that, in spite of the great increase in general interest among both surgeons and physicians, there is little standardization in the form of therapy. Of chief interest to us is the

*From the Surgical Department of the Peter Bent Brigham Hospital and the Harvard Medical School.

Read before the Nineteenth Annual Meeting of the American Association for Thoracic Surgery, May 4, 5, 6, 1936.

opportunity to compare the results of medical and surgical therapy, partly because of the insight it gives us into the fallacies of statistical evidence. An intimate study of the case histories in the group we shall present makes it obvious that if all our abscesses of the lung were submitted to surgery, our mortality rate would have been considerably cut down. But in doing this we would have submitted to surgery a group of patients who would have recovered spontaneously. Thus it seems certain that the man who believes all cases of pulmonary abscess should have surgical therapy will have a lower mortality rate than his colleague who tries to set certain cases aside and centers his energies upon those who have failed to respond to medical therapy.

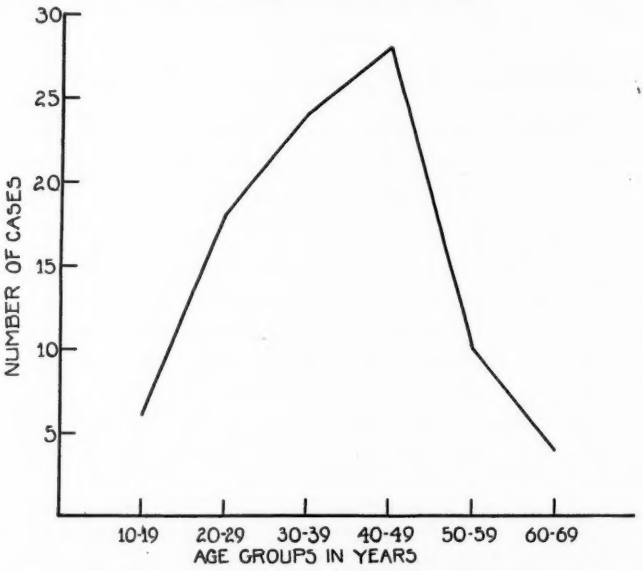

Fig. 1.—Graph showing the incidence of lung abscess according to age groups; in a series of 90 cases.

We wish to report upon all cases of abscess of the lung treated on the medical and surgical services of the Peter Bent Brigham Hospital during the twenty-year interval, 1914 to 1934. Eighty-five patients (95 per cent of the series) have been followed for periods varying from two to twenty-two years so that it is possible to estimate satisfactorily the end-results of therapy. The study is confined to patients with localized pulmonary abscess and does not include consideration of spirochetal gangrene, actinomycosis, primary bronchiectasis, or miliary abscesses developing as a result of bacteremia.

Age Distribution.—Lung abscess does not appear to be restricted to any age group, and the incidence in our patients corresponds roughly to the greater number of middle-aged people we see on the general sur-

gical and medical services. Indeed, the incidence is exactly comparable to the incidence of postoperative pulmonary complications as a whole.[5] Inasmuch as children are not treated at the Brigham Hospital, we had no experience with pulmonary abscess in the young. Separated into the decades of life, we find the distribution of abscesses as indicated in Table I and Fig. 1.

TABLE I

AGE DISTRIBUTION OF LUNG ABSCESS

AGE IN YEARS	TOTAL NUMBER OF CASES
10-19	5
20-29	18
30-39	24
40-49	28
50-59	11
60-	4

Sex Distribution.—Sixty-one of the patients were males and 29 were females, making an incidence in men about twice as high as that in women. This agrees with the general statistics for pulmonary disease; for example, McCrae,[25] in a study of 12,098 cases of lobar pneumonia, found that 73.4 per cent occurred in males. Whether this is related to the different types of respiration in the two sexes, thoracic in women and diaphragmatic in males, or to other etiologic factors, we have no evidence to determine.

PATHOGENESIS

The etiology of nontuberculous pulmonary abscess is a subject of much discussion and forms the basis for extensive laboratory investigation. Whether an abscess in a given case originated from material introduced by way of the bronchial tree, or whether it resulted from embolism and infarction, cannot be determined with certainty except in a small percentage of cases. Clinicians may interpret the early symptoms and signs as due to embolism or aspiration according to their general beliefs, and the pathologist only rarely sees a case in the early phase of the disease when the etiologic mechanism can be accurately reconstructed. So much has been written supporting the various theories of pathogenesis that the resulting maze is likely to bewilder the clinician and make him believe that the question is only of academic interest. In spite of the confusion that has resulted, it still remains clear that clinical correlation and laboratory study must continue, for only by finally settling the question of etiology can we hope to direct intelligent efforts toward the prevention of this serious malady.

In reviewing the following group of 90 cases we find that 54 per cent developed after a surgical procedure, 28 per cent after pneumonia, 4 per cent after bronchiectasis; whereas in 14 per cent of the cases there arose insidiously vague symptoms which left us in doubt concerning the

true etiology. These figures are listed in Table II. The percentage of postoperative cases is distinctly higher than is usually reported in the literature, for a previous review of 1908 collected cases[6] showed 29 per cent occurring after operation and 14 per cent of the total after tonsillectomy.

TABLE II

ORIGINATING CAUSES OF PULMONARY ABSCESS

ETIOLOGIC FACTORS	NUMBER OF CASES	PERCENTAGE OF SERIES	
Tonsillectomy	22	24%	54%
Teeth extraction	13	14%	
Other operations	14	16%	
(Biliary 3, gastrectomy 2, herniorrhaphy 2, appendectomy 2, hemorrhoidectomy 2, nasal 2, cerebellar 1)			
Pneumonia	25	28%	
Bronchiectasis	3	4%	
Etiology unknown	13	14%	

In the postoperative cases of this series it is impossible in most instances to find clean-cut evidence in support of aspiration or embolism as the responsible etiologic mechanism. In an occasional case (such as No. 25) there was fairly good evidence that pulmonary embolism was the most important causative factor, but even here we must admit that infarction might have been accompanied by aspiration of mouth organisms in sufficient numbers to be an important consideration. It is rather impressive to find, in an analysis of the postoperative course of patients who later developed an abscess, some instances in which there was either a difficult induction period or trouble because of the collection of secretion in the pharynx and trachea or struggling when the narcosis was not deep enough. These examples tend to make one believe that aspiration of mucus, blood, or débris (from tonsillectomy) might well have been the inciting cause of the abscess; yet it is a fact that expert anesthesia does not greatly reduce the incidence of this postoperative complication, and the condition occurs even after the use of local anesthesia either for tonsillectomy or for other operative procedures.

A study of the distribution of the abscesses according to pulmonary lobes seems to us to throw some light on the mechanism involved in the development of the lesion. If pulmonary abscess resulted commonly from aspiration of blood or infected material, it is logical to expect that a high proportion of the cases would show involvement of the right lower lobe; for, while material entering the bronchial tree could theoretically have access to any part of the lungs, the larger passageway and the straighter course to the right lower lobe would tend to lead most foreign substances in this direction. A glance at Table III, however, does *not* show any preponderance of abscesses in the right lower

lobe. In fact, and in agreement with other reporters, abscess of the lung in this (post-tonsillectomy) group showed a greater distribution in the upper lobes.

If embolism were the important factor in production of lung abscess, one might expect to find the lesions widely throughout the lung fields. It is common experience at the autopsy table to find aseptic infarcts (unassociated with abscess formation) in diverse portions of the lung, and the pathologist is accustomed to seeing small isolated emboli in almost any portion of the pulmonary vascular bed. If such emboli were infected or if organisms were present in the lung tissue which might cause necrosis in these areas of infarction, abscess formation would probably result. Therefore we are prone to believe that the widely scattered distribution of lung abscesses as shown by Table III is rather in favor of the embolic theory of their pathogenesis in many instances.

TABLE III

DISTRIBUTION OF ABSCESSES

ETIOLOGY	LOBE INVOLVED				
	RIGHT UPPER	RIGHT MIDDLE	RIGHT LOWER	LEFT UPPER	LEFT LOWER
Tonsillectomy	6	1	3	9	3
Teeth extraction	3	1	7	2	0
Other operations	6	0	2	1	5
Pneumonia	7	1	5	9	3
Bronchiectasis	1	0	2	0	0
Unknown etiology	3	2	2	1	5
Totals	26	5	21	22	16

It is an unfortunate circumstance that clinicians are seldom able to study by x-ray examination the development of an abscess from its early stages. Conditions are naturally such that the individual usually does not present himself until pathologic changes are well advanced in the lung. If it were possible to study carefully by roentgenography a group of individuals through the incipient phases of their disease, it is quite probable that additional information would be gained concerning the etiologic processes. Thus, the earliest x-ray abnormality might prove to be that of infarction, or the character of the pulmonic lesion might indicate atelectasis from bronchial occlusion, and in either instance give definite support to the embolic or aspiration theory respectively. One occasionally does see indisputable evidence of embolism. Such is seen in the case of L. M., a married woman of twenty-six years, with classical cholelithiasis. Uneventful cholecystectomy and cholecystostomy were performed under nitrous oxide and ether anesthesia. Recovery was uncomplicated until the ninth postoperative day when there was sudden onset of pain with respiratory movement beneath the right clavicle. Temperature was elevated to 39.8° C. and remained elevated for three days. There was slight cough but no sputum. Roentgenograms showed

a triangular induration in the axillary region. Twenty-nine days after the operation there was definite x-ray evidence of an abscess in the triangular shadow with a fluid level. By the thirty-ninth postoperative day all evidence of pulmonary pathology had disappeared. The form of the indurated area in the roentgenogram and the absence of sputum seem to us to suggest embolism as the etiologic factor in this case.

In some postoperative cases there is evidence that the earliest signs and x-ray findings are those of bronchopneumonia, which in the presence of a particular bacterial flora goes on to abscess formation. This appears to be a tenable explanation of what may happen in some postoperative cases, for certainly this series of events obtains in patients who have *not* been operated upon, as is clearly shown by the 23 patients (Table II) in whom abscess developed following a pneumonia. In such postoperative pneumonia cases it is possible that necrosis occurs by the action of organisms such as the Friedlander's or Vincent's or Melaninogenicum type.

In reviewing the conditions which are likely to be present in a lung before abscess develops, it is necessary to bring up the subject of spirochetal pulmonary gangrene. Without doubt this process may have widespread distribution in pulmonic tissue and yet may heal completely with proper medical care and arsenical medication. We are convinced, however, that some of these individuals develop large abscesses of either a localized or a multilocular variety, and we are impressed by the fact that pulmonary gangrene and abscess cannot always be separated into two distinct entities as Kline and Berger[19, 20] would have us believe. Therefore, while we acknowledge the excellent work which these authors have done in establishing spirochetosis as an important disease, we still recognize that in some cases this condition is closely related to the development of a pulmonary abscess.

It is impossible, because of insufficient space, to summarize and comment upon the experimental production of pulmonary suppuration which various authors have previously set forth. Reviews and discussions of this aspect of the problem have been made by Heuer,[13] Lambert and Weeks,[21] Cutler,[8] and others. Suffice it to say that after considerable difficulty Crowe and Scarff[4, 27] were able to produce occasional acute lung abscesses by instillation into a bronchus of foreign bodies infected with material collected from chronic nasal suppuration or from carious teeth. Opposed to this, an abscess of the lung could be readily produced in dogs by Cutler and Schlueter,[6] and Holman, Weidlein and Schlueter[15] by releasing infected emboli into the venous circulation. If the infecting organism was one of the ordinary pyogens such as *Staphylococcus aureus,* the abscess tended to heal quickly; but, if more virulent and anaerobic bacteria were employed (such as obtained from the mouth), the abscess was usually larger and more chronic.[12] Also it could be shown that there was a definite relation between the type of pulmonary

lesion produced and the preexisting immunity which had been built up in the animal.[6] Van Allen and his workers[31, 32] have taken a middle course and could demonstrate that embolism with an infected or aseptic clot was the primary factor in producing local tissue injury in the lung but that the important organism producing chronicity probably gained access to the lung by way of the bronchial tree.

TREATMENT

In studying our cases we have listed as *cured* those who have shown complete healing of the cavity by x-ray examination and who have no residual symptoms. A small number have been classified as *improved* because there has been some persistent mild symptom such as a cough or incomplete clearing by x-ray, but in all these patients there has been a return to good general health and the results of treatment have been satisfactory. In the *unimproved* class the clinical course or healing by x-ray has not been satisfactory or the symptoms have become worse.

In this series of 90 cases, *medical treatment* alone was employed in 43. Of this number 38 have been followed so that the end-results of therapy are available. These are summed up in Table IV.

TABLE IV

RESULTS OF MEDICAL TREATMENT

Patients	Cured	20	Satisfactory results	70%
	Improved	7		
	Unimproved	1		2%
	Dead	10	Mortality	28%

The end-results of medical treatment are slightly better than the mortality figure of 28 per cent would at first indicate, for included in the 10 deaths were two patients (one with uremia and one with a cerebellar neoplasm) in which the complicating condition was largely responsible for the fatal issue. If these two cases are excluded from the series, the mortality rate of medical treatment in cases of lung abscess in which there were no other complicating disease would be about 25 per cent.

In the series of 90 cases, *surgery* was employed at some time during the treatment of 47 patients, the results of which are summarized in Table V.

TABLE V

RESULTS OF SURGICAL TREATMENT

Patients	Cured	16	Satisfactory results	47%
	Improved	6		
	Unimproved	3		8%
	Dead	22	Mortality	45%

At first sight it would seem that the mortality in the surgically treated patients is appallingly high as compared with the results of med-

ical therapy, but it should be noted that included in this surgical mortality rate are deaths from complicating causes which do not appear in the medically treated group. Thus, there were seven patients who had an empyema or pyopneumothorax, and this complicating factor was largely responsible for death. If these seven cases were excluded, the adjusted mortality rate for surgical treatment would be approximately 33 per cent. This, of course, is still high when contrasted to 25 per cent for medical treatment of uncomplicated cases, but the mortality statistics for the two forms of treatment should not be compared, since different types of cases are selected for medical or surgical therapy. It is not surprising that the surgical mortality is relatively high, for we must accept as operative risks many patients who failed to progress satisfactorily on medical treatment.

It is true that, when we break down our twenty-year interval into five-year periods, it is apparent that surgical therapy is becoming a little less hazardous and giving more satisfactory results. In the period 1915-1919, 27 per cent of the cases were satisfactory and 73 per cent unsatisfactory or dead; in the last five-year period, 1930-1934, 66 per cent were satisfactory and only 34 per cent unsatisfactory or dead. Moreover, in this last group were two cases in which cautery pneumonectomy on a large scale was attempted, both cases resulting fatally. It was this experience that led us to discard, except in very unusual circumstances, this method of treatment.

Medical therapy in the above listed cases had as its mainstay postural drainage of conventional type: namely, the placing of a patient in such a position as to promote better evacuation of the cavity by the gravity method. Depending upon the lobe involved, the patient would be placed leaning head down over the side of a bed, resting on one side or the other, maintaining such a position for fifteen to twenty minutes three or four times a day. In this way 200 c.c. to 400 c.c. of purulent exudate were commonly brought up a day, one patient raising as much as 800 c.c. of sputum a day. The amount of drainage did not prove to have any prognostic significance, for one individual might expectorate alarming quantities of pus and recover completely, whereas another patient would cough up decreasing amounts of sputum and become progressively more sick because he did not have a free drainage from the abscess cavity.

In addition to postural drainage, a good medical regime included supportive symptomatic treatment, sedatives when required, expectorants as needed, a nourishing diet, transfusions when necessary, and as much fresh air and sunlight as was possible to give. Calcreose (4 c.c. three times a day) was prescribed for most of our patients because of its beneficial effects in loosening up secretions and easing the cough. Hemoptysis required the same emergency treatment as in pulmonary tuberculosis, and while we have had severe bleeding of 400 c.c. to 600 c.c. in a few of

our cases, we have had only one case of fatal hemorrhage. Arsphenamine in doses of 0.4 gm. was given as often as three times a week to those patients who had spirochetes and fusiform bacilli in their sputum (about 80 per cent of the series). In no case did we find any striking benefit from the use of this drug, yet it was commonly administered in the hope that it might exert some favorable influence on the course of the disease.

Fig. 2. Fig. 3.

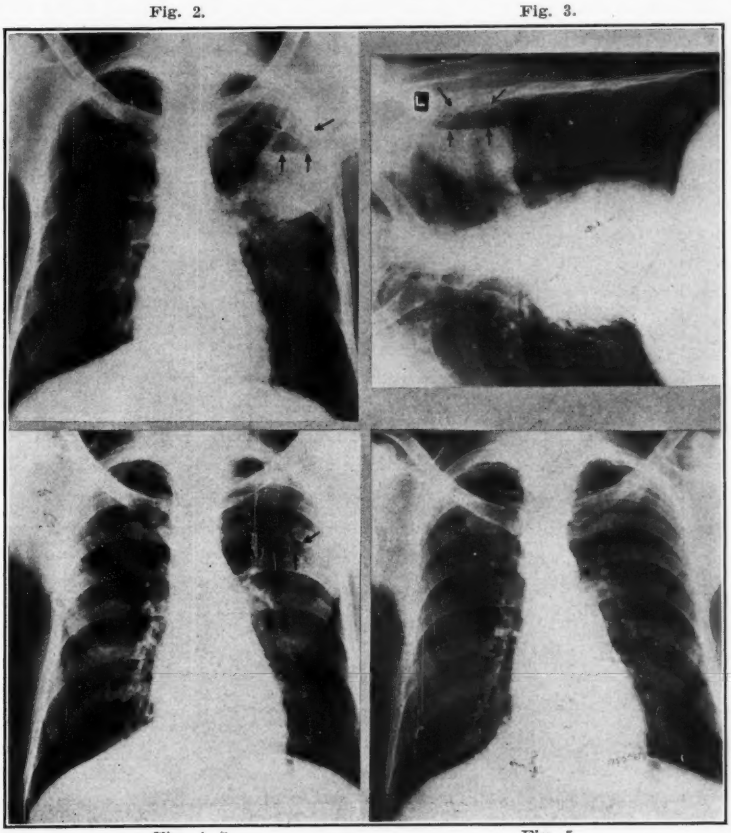

Fig. 4. Fig. 5.

Fig. 2.—Case 26. Large abscess of left upper lobe, showing fluid level; patient in erect position.

Fig. 3.—Case 26. Same as Fig. 2, patient lying on side.

Fig. 4.—Case 26. X-ray three months after Figs. 2 and 3, showing progress in healing of cavity on medical treatment.

Fig. 5.—Case 26. X-ray six years after Figs. 2, 3 and 4, showing end-result and complete healing of lung abscess.

Collapse therapy by induction of artificial pneumothorax has fallen somewhat into disfavor. Ten years ago it was employed with considerable enthusiasm in many of our cases, but the disappointing results obtained have made us abandon the procedure. It is difficult to explain

why the method should not promote better healing of an abscess, and the only logical explanation which seems tenable is that the collapse of the lung reduces the size of the aperture leading from abscess to bronchus and thereby causes retention of exudate in the abscess cavity. Besides the fact that the procedure lacks therapeutic efficacy, there is an attendant danger of producing an empyema or pyopneumothorax which may prove difficult to treat. Such accidents are often the turning point from

Fig. 6. Fig. 7.

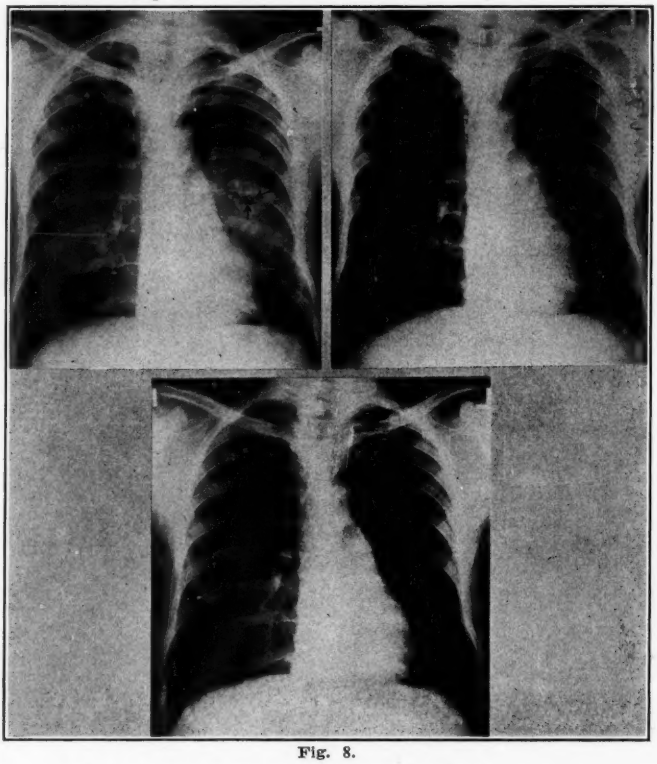

Fig. 8.

Fig. 6.—Case 21. Small, centrally located abscess on left.

Fig. 7.—Case 21. Diminution in size of abscess with postural drainage. Film taken three weeks after Fig. 6.

Fig. 8.—Case 21. Abscess completely healed. Film taken two months after Fig. 6.

a favorable outlook to a poor prognosis, and these dangers should discourage the use of artificial pneumothorax in the treatment* for lung abscess.

*Occasionally pneumothorax may be of value in diagnosing abscess at the very bottom of a lower lobe, the collapse elevating the legion from behind the diaphragm so that it may be studied roentgenologically. It has also been used by some to study pleural adhesions. When used for the above reasons only moderate collapse need be attained.

The *surgical measures** for treating pulmonary abscess involve the use of five or six different procedures, selecting the type of operation which the individual case demands. The most important single procedure is the evacuation of the abscess cavity by simple external drainage in one or two stages. This was carried out in 33 of the 47 cases. In 26 instances the parietal and visceral pleurae were found to be adherent to

Fig. 9. Fig. 10.

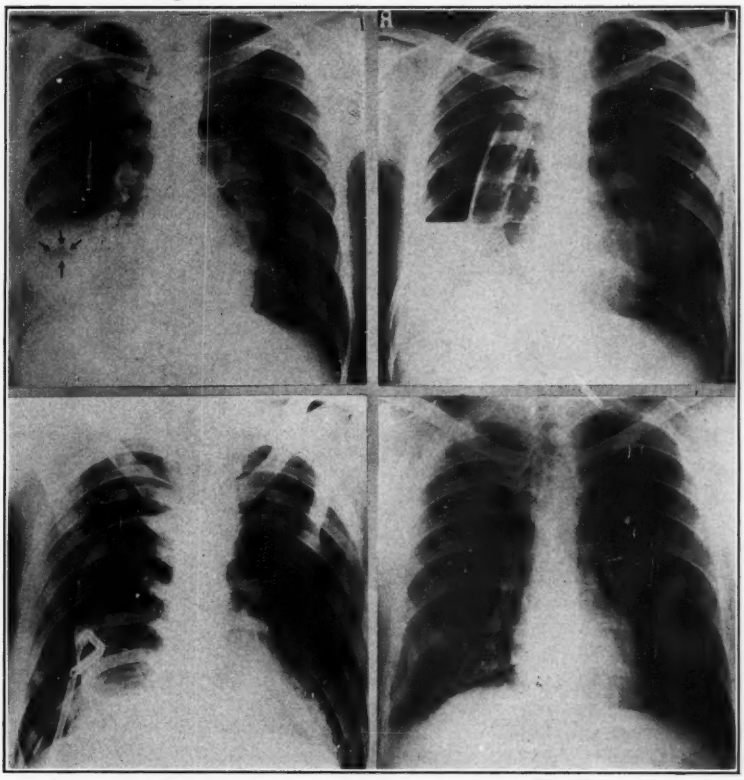

Fig. 11. Fig. 12.

Fig. 9.—Case 85. Abscess of right lower lobe, with extensive pneumonitis surrounding it.

Fig. 10.—Case 85. Spontaneous rupture of abscess with development of pyopneumothorax and collapse of lung.

Fig. 11.—Case 85. Drainage of pleural cavity by catheter. (Rib resection done later when fluid reaccumulated.)

Fig. 12.—Case 85. End-result (two and one-half years after Figs. 9, 10 and 11).

each other and permitted immediate entrance to the lung without danger of pulmonary collapse or empyema formation. In the remaining

*The majority of the cases in this series, outside of the authors' own cases, were operated upon by Drs. John Homans and H. F. Newton, although all members of our surgical staff, including past resident surgeons, have handled a few of the patients covered in this report. Dr. Homans[16] has previously reported 23 of the cases herein studied, and Dr. Wood[26] has recently published the pyopneumothorax material.

seven patients, the two pleural surfaces were found to be nonadherent, and a two-stage operation was necessary, sewing the two pleural layers together at the first operation and then reopening the wound to drain the abscess at a second sitting. Formerly it had been common practice at the first stage to remove sections of the ribs at the desired site and then to pack the wound with gauze in the hope that sufficient foreign body reaction would be called forth to make the adjacent visceral pleura seal itself to the parietal one. We have had disappointments with this,

Fig. 13. Fig. 14.

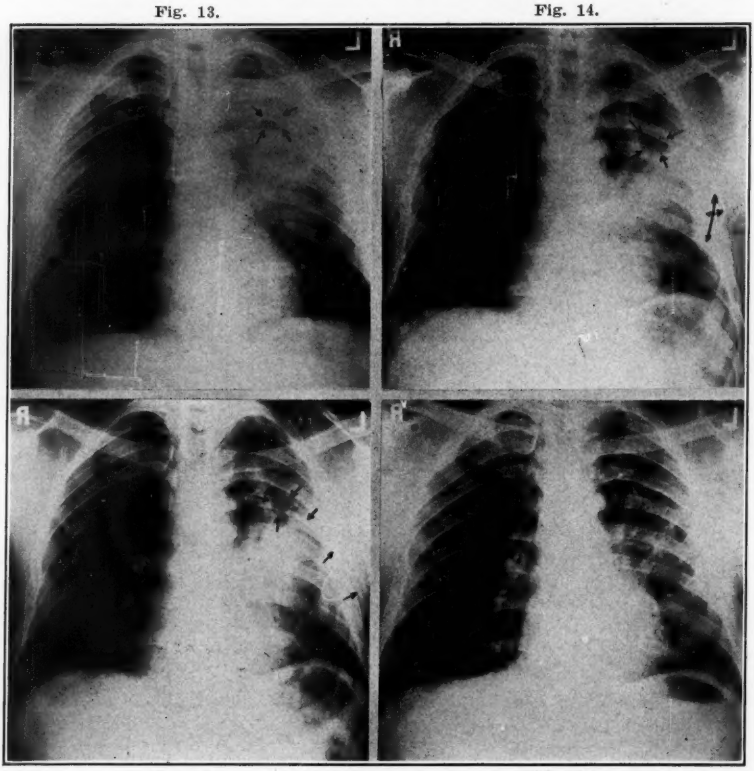

Fig. 15. Fig. 16.

Fig. 13.—Case 86. Abscess of left upper lobe, with surrounding pneumonitis.

Fig. 14.—Case 86. Film seven months after Fig. 13. On medical treatment the abscess had healed and recurred. Lower arrows point to silver wire left in wound at first-stage operation (to show relationship of wound to lung abscess).

Fig. 15.—Case 86. Film taken after second stage of surgical drainage of abscess. The three arrows toward the right point out the catheter entering the lung abscess above.

Fig. 16.—Case 86. Follow-up film two years after Figs. 14 and 15. Fibrosis of lung is present, with complete healing of the abscess cavity. (Patient clinically cured.)

however, finding at the second operation two or three weeks later that the two layers were still ununited. Therefore, it is better practice to sew the two layers together at the first operation. This can be readily ac-

complished by removing sections of the ribs and (without opening the pleura) taking stitches which catch both layers and fasten them together. In addition to this, the wound may be packed with gauze. The anesthetic of choice is local novocaine infiltration of the chest wall at the operative site. A general anesthetic should be avoided because the operative manipulations may set up pulmonary bleeding or may release purulent exudate into the bronchial tree which could not be expectorated if the pa-

Fig. 17. Fig. 18.

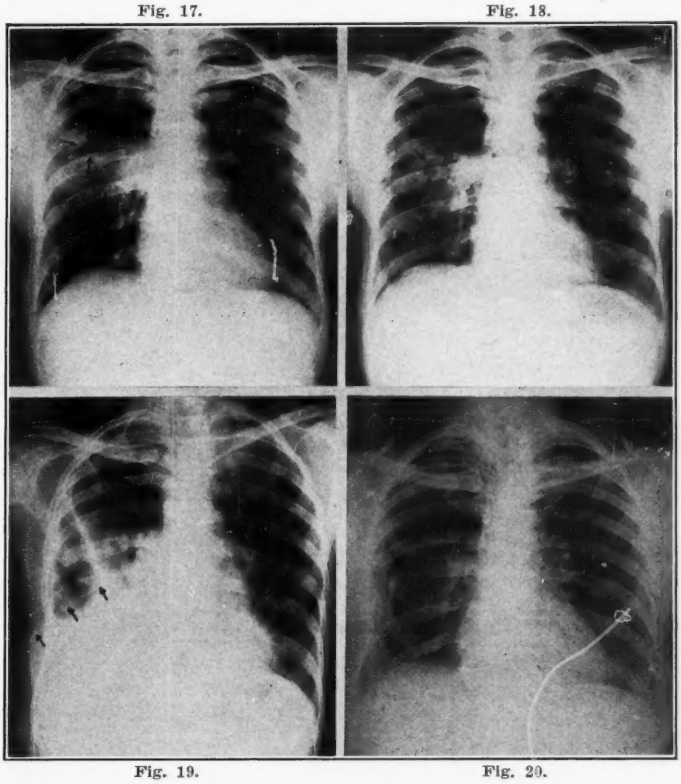

Fig. 19. Fig. 20.

Fig. 17.—Case 84. Abscess of right lung, with moderate amount of peripheral pneumonitis.

Fig. 18.—Case 84. Slight diminution in size of abscess and in surrounding reaction after postural drainage. Film taken six months after beginning of abscess and two months after Fig. 17.

Fig. 19.—Case 84. Surgical drainage of abscess. Film taken seven months after Fig. 17. Arrows point to drainage tube.

Fig. 20.—Case 84. End-result, three years after Fig. 19. Patient clinically cured.

tient is unconscious or if the cough reflex is abolished. Incision of the lung should be done with an endothermy knife if the abscess has a peripheral location; but, if it has a central position, the actual cautery should be employed in order to lessen the danger of hemorrhage from large vessels of the hilum.

After external drainage of an abscess has been accomplished, it is not always an easy matter to keep this sinus tract patent as long as is desired. The opening through the chest wall, and even the lung tissue, has a strong tendency to close down; and while it may not heal over completely, it at least interferes with proper drainage. We have felt that vertical incisions rather than incisions parallel to the ribs tend to stay open longer and permit better drainage without a tendency toward the pocketing of infection under the skin flaps. It may be necessary to enlarge the fistulous tract from time to time as the circumstances demand and as drainage is needed. Of the 33 cases in which drainage was done, it was necessary in six to perform subsequent operations (one to four) for enlarging the opening in either the lung tissue or the thoracic wall.

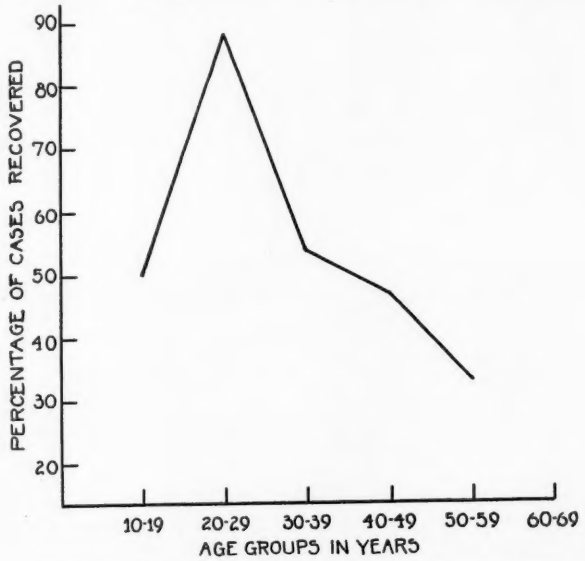

Fig. 21.—Graph showing percentage of recoveries from lung abscess in various age groups. In the age periods of twenty to twenty-nine years, 88 per cent of the patients were cured or satisfactorily improved.

Next to the direct drainage of a pulmonary abscess, the most important operative procedure is concerned with the treatment of a complicating empyema or pyopneumothorax. Invasion of the pleural space by infection from a pulmonary abscess usually constitutes a surgical emergency, for in most of these cases there is the sudden rupture of an abscess with the discharge of purulent exudate into an undefended space, and this is usually accompanied by the formation of a bronchopleural fistula. This series of events leads to a rapid increase in the toxicity of the patient, accompanied by embarrassment of the respiratory and circulatory systems, because of collapse of the lung and mediastinal flutter. Furthermore, a positive pressure, tension pyopneumothorax may develop

and make relief even more imperative. Empyema may be treated by the same measures as are instituted for acute empyema occurring as a complication to pneumonia; and the choice of closed drainage, open tube drainage, or open thoracotomy by rib resection will again depend upon the duration of the empyema, the amount of mediastinal fixation and the viscosity of the exudate which is present. Pyopneumothorax, however, should never be treated by aspiration or closed drainage because

Fig. 22. Fig. 23.

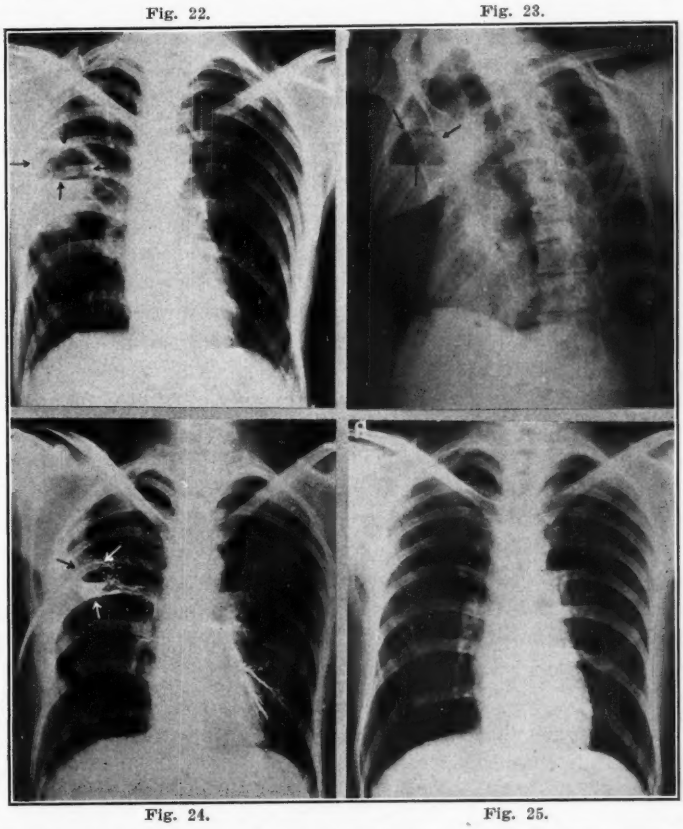

Fig. 24. Fig. 25.

Fig. 22.—Case 88. Large abscess of right upper lobe, with little surrounding reaction.

Fig. 23.—Case 88. Same as Fig. 22, left anterior oblique view, showing abscess to be in anterior portion of the lung.

Fig. 24.—Case 88. Film taken three months after Figs. 22 and 23, following two months of medical treatment and one month of surgical drainage. Lipiodol injected through catheter shows fluid level in abscess, with communication to bronchial tree. Arrows outline approximate size of cavity.

Fig. 25.—Case 88. Film of chest one year after Fig. 24, showing complete healing.

these measures will promote further discharge and sucking of exudate from the abscess into the pleural cavity. The treatment of pyopneumothorax then resolves itself into either the rapid insertion of an open tube

into the chest when the individual is quite toxic or the resection of a rib if the patient is able to stand this procedure and the exudate is thick enough to require the larger opening. The number of patients (twelve) which we have operated upon for empyema and pyopneumothorax is too small to permit final deductions as to the advantages of any special procedure, but we feel warranted in making the above generalizations.

Although drainage of the abscess or a complicating empyema is the chief procedure in this field, occasionally it is necessary to remove or destroy diseased lung tissue. This is best performed by the cautery pneumectomy method of Graham. A simple pulmonary abscess is sometimes accompanied by or is followed by an extensive necrosis of nearby pulmonic parenchyma, which condition can best be described by the term "chronic pulmonary suppuration." Such a process is often very resistant to treatment by conservative means, and the interests of the patient are best served by actual removal of the substance thus involved. We have employed this operation in six cases in the present series. After the lung is once exposed, the individual stages of removal of the lobe can be done without anesthesia, charring and burning away as much lung at each sitting as appears to be safe. With this operation we have obtained clinical improvement in three of our six patients thus treated. However, two of these still have bronchocutaneous fistulas. In view of the widespread and increasing use of the endothermy knife, it is well to caution against its employment in removing lung tissue. It has been our sad experience to use it in one case in which a pulmonic vein was opened. As the patient inspired, there was a gush of air into this opened vessel with a resultant death in three or four minutes from extensive air embolism. Therefore, the soldering iron or the actual cautery, as originally proposed by Graham, is still the best instrument for burning away, sealing and charring the exposed lung surface in such a way as to minimize this hazard of fatal embolism.

Thoracoplasty deserves little mention in the treatment of nontuberculous pulmonary abscess, for its high success in the treatment of cavitation as seen in tuberculous lesions is no criterion for its use in the cases here discussed. While analogous conditions would at first make it appear that collapse therapy might promote obliteration and healing of a cavity, experience has shown that the results obtained do not support this reasoning. We have employed partial thoracoplasty on three occasions, one of which was in conjunction with cautery lobectomy, but the results in this limited experience lead us to discourage its use.

Phrenicectomy would seem to be of some value in the treatment of abscesses situated in either lower lobe, but again the empirical results did not fulfill the expected gain, and we have abandoned the procedure in treating this type of pulmonary abscess.

Bronchoscopy has a definite place in the care of these patients, and each one, if not too ill, should have the benefit of this investigation to rule out the possibility of a foreign body as the inciting cause of the malady. In the hands of one who is adept at the use of the instrument, bronchoscopy produces little or no untoward reaction and occasionally will give valuable information regarding the presence of a neoplasm, foreign body or bronchial stricture which must necessarily receive attention if the abscess is to be treated properly. We did not examine as many of our patients in this way as we should have, but we now advocate its more extensive use. In a few cases we have done repeated bronchoscopies for aspirations of abscess exudate but have found that, if a sufficient opening already exists between abscess and bronchus to permit this type of treatment, the patient would probably do as well with postural drainage alone. Therefore we do not advise the use of the bronchoscope for repeated aspirations, but do recommend it for its value as a diagnostic aid.

PROGNOSIS

The type of condition giving rise to a pulmonary abscess has little bearing on the prognosis and the chances for successful issue. If the cases be grouped according to etiologic conditions as shown in Table VI, it will be seen that in each division there are about the same proportion of favorable cases (cured and improved) as unfavorable ones (unimproved and dead). The only important exception to this is that group in which the abscess had a vague onset and developed from an unknown cause. While the number of patients in this latter group is too small to warrant any conclusions, yet the impression is gained that in this division the prognosis is poorer than in the series as a whole. In this group there was often a greater amount of chronic pneumonitis about the abscess than was found in other cases.

TABLE VI

RELATIONSHIP OF ETIOLOGY TO PROGNOSIS

ETIOLOGY	FAVORABLE CASES (CURED AND IMPROVED)	UNFAVORABLE CASES (UNIMPROVED AND DEAD)
Tonsillectomy	12	8
Teeth extraction	10	3
Other operations	6	7
Pneumonia	17	7
Bronchiectasis	1	2
Unknown etiology	3	9

The age of the patient is one of the most important considerations in the estimation of prognosis. As one might expect, young adults have a much more favorable outlook than do other age groups and in the period

of twenty to thirty years the percentage of recoveries reaches a high figure of 88 per cent, in sharp contrast to other age decades. These are listed in Table VII and are shown in Fig. 21.

TABLE VII

RELATIONSHIP OF AGE TO PROGNOSIS

AGE OF PATIENT	FAVORABLE CASES (CURED AND IMPROVED)	UNFAVORABLE CASES (UNIMPROVED AND DEAD)	PERCENTAGE OF RECOVERIES
10-19 years	3	3	50%
20-29 years	15	2	88%
30-39 years	14	9	60%
40-49 years	13	15	47%
50-59 years	3	6	33%
60 years	2	1	--

The position of an abscess in regard to the various pulmonary lobes has no importance as far as the prognosis is concerned. Table VIII lists the distribution of the lesions in our cases and shows the corresponding favorable or unfavorable results of therapy. It appears that in this series of cases an abscess in the right lower or the left upper lobe had a better chance of successful issue as contrasted to one situated in the left lower lobe. With a greater number of cases this relationship might not hold true.

TABLE VIII

RELATIONSHIP OF PROGNOSIS TO POSITION OF ABSCESS

LOBE INVOLVED	FAVORABLE CASES (CURED AND IMPROVED)	UNFAVORABLE CASES (UNIMPROVED AND DEAD)
Right upper	13	10
Right middle	2	3
Right lower	14	7
Left upper	14	6
Left lower	6	10

The presence of a complicating empyema or pyopneumothorax of course alters the outlook in a given case. Fourteen of our patients developed one or the other of these processes (nine empyema and five pyopneumothorax) and of these eight died. This gives a mortality of 57 per cent in the 14 cases with infection of the pleural space in contrast to a total mortality of 38 per cent in the remaining cases which did not have this condition. In at least two of our patients the pleural infection occurred as a direct result of operative procedure, attempted drainage of a pulmonary abscess when the visceral and parietal pleurae were not sufficiently adherent. In several patients an abscess situated in the peripheral portion of the pulmonary parenchyma spontaneously ruptured into the pleural cavity. In the great majority of cases, however, the development of an empyema or pyopneumothorax appeared to be

the direct result of injudicious chest tapping, and it must be pointed out that this needling may be fraught with considerable danger.

The length of illness from the onset of symptoms until hospitalization and institution of treatment bears some relation to the prognosis, but not so much so as one might expect. It is surprising to find that an abscess of many months' standing may heal as completely and as promptly as another of only two months' duration. The state of affairs may be summarized by saying that if treatment is instituted during the first month of the disease, the chances of recovery are better than if the condition is left untreated for a longer period. However, after two months' duration the prognosis does *not* necessarily become worse merely because the abscess has been of long standing. Table IX sums up the favorable and unfavorable results in our patients with relation to the duration of symptoms before hospitalization and institution of treatment.

TABLE IX

RELATIONSHIP OF PROGNOSIS TO DURATION OF ABSCESS

DURATION OF ABSCESS PRIOR TO TREATMENT	FAVORABLE CASES (CURED AND IMPROVED)	UNFAVORABLE CASES (UNIMPROVED AND DEAD)
1 month	24	14
2 months	7	7
3 months	3	4
4 months	4	1
5 months	1	0
6 months	0	2
6 months plus	10	8

In concluding a discussion of the prognosis in nontuberculous pulmonary abscess, it should be emphasized that these patients ought to be treated for a considerable period of time on a regime which is as rigid as that which is universally prescribed for pulmonary tuberculosis. These individuals may show such great improvement after the institution of postural or surgical drainage that at the end of one to three months they are inclined to discontinue further treatment, believing they are cured. It has been a disappointing experience to see some of them regain almost normal health and then relapse into their former condition and, in some instances, die two or three years later. It is often impossible to provide adequate hospitalization in a unit which is organized for the treatment of acute surgical or medical cases, and it is frequently a difficult problem to find suitable institutions where prolonged care can be obtained. Tuberculosis sanatoriums are usually closed to them, and nursing homes will seldom give the proper rest, inactivity or fresh air and sunshine therapy which these people need. It is all too easy to discharge them to their homes after the alarming or disagreeable symptoms of their disease have passed, but one of the most important considerations in assuming responsibility for an individual of this kind is to arrange for an adequate convalescence and to follow him or her closely over a period of several years.

SUMMARY

To prevent the development of pulmonary abscess after surgical operations, it is necessary to recognize the various factors which might be important in causing this condition. Operative manipulation must be gentle, and mass ligatures should be avoided in order to reduce the hazard of pulmonary infarction by embolism. Adequate oral hygiene must be instituted before operation is undertaken, whether it is to be done under novocaine or general anesthesia, so that the danger of infecting the bronchial tree is reduced to a minimum. In the presence of a postoperative bronchopneumonia, secondary infection of the lung tissue should be guarded against by proper cleansing of the teeth, gums and pharyngeal cavity. In order to prevent the development of abscess in patients who have not been subjected to surgical procedures, every case of pulmonary disease (pneumonia, bronchiectasis, etc.) should have especial attention directed toward prevention of superimposed invasion by the anaerobic "mouth organisms."

In all cases of pulmonary abscess, medical treatment with postural drainage and supportive measures should be instituted for a period of at least six weeks. If progress in the healing and drainage of the cavity is not evident in this period, operative drainage should be instituted. Of 38 cases followed after treatment by medical means there were satisfactory results in 68 per cent and a fatal outcome in 29 per cent. The use of arsphenamine or neoarsphenamine appeared to have little value in treating those cases in which spirochetes were present in the sputum.

The use of artificial pneumothorax for collapsing a pulmonary abscess should be discouraged, for the procedure often has little curative value and is dangerous because it may induce the complication of empyema or pyopneumothorax.

In the surgical treatment of 47 patients with abscess, satisfactory results were obtained in 47 per cent of the cases and there was a total mortality of 45 per cent. Excluding cases treated for a complicating empyema or pyopneumothorax, the mortality rate for surgical treatment of the pulmonary abscess alone was 33 per cent.

In 33 cases in which attempts were made to drain the abscess surgically, it was possible to perform a one-stage procedure in 26; but in the remaining seven cases the pleural surfaces were not adherent, and a two-stage operation was necessary.

Thoracoplasty has little place in the treatment of pulmonary abscess except when it is desired to combine this procedure with cautery pneumectomy.

Various factors relating to the prognosis in a given case are discussed. It is essential in treating individuals with this condition that they be followed for a period of several years before they can be assured that a complete cure has been effected, and during this interval they should be held to a hygienic regime similar to that set up for patients with pulmonary tuberculosis.

DATA IN TREATED CASES OF LUNG ABSCESS

CASE	AGE	SEX	ETIOLOGY	DURATION OF ABSCESS	LOBE INVOLVED	SIZE OF ABSCESS	TREATMENT	RESULT
1	36	M	10 days after tonsillectomy	2 mo.	Right lower	8 cm. diam.	Postural drainage 3 months	Cured
2	25	F	? Postpneumonic	1 mo.	Left upper (developed empyema)	6 cm. diam.	Postural drainage 3 months (aspiration of empyema)	Cured
3	66	M	Postoperative pneumonia	1 mo.	Right upper	3 cavities 3 cm. diam. 2 cm. diam. 1 cm. diam.	Postural drainage 3 months	Died
4	22	F	1 month after tonsillectomy	3 mo.	Left upper	2 cm. diam.	Postural drainage. Repeated bronchoscopies	Not followed
5	49	M	? Postpneumonic	4 mo.	Right upper		Postural drainage 1 month	Improved
6	64	M	Postpneumonic	1 mo.	Left upper	Irregular, multilocular	Postural drainage 1 month	Improved
7	34	M	? Postpneumonic	1 mo.	Right lower	3 cm. diam.	Postural drainage 6 weeks (diagnostic bronchoscopy)	Improved
8	42	M	1 month after cerebellar operation; postoperative pneumonia	1 mo.	Left lower	8 cm. diam.	Postural drainage 4 weeks	Died in 4 weeks
9	47	M	3 weeks after tooth extraction	11 mo.	Right lower	2.5 cm. diam.	Postural drainage 3 months. Bronchoscopy	Cured

NOTE.—Cases 1 to 43 treated medically. Cases 44 to 90 treated surgically.

DATA IN TREATED CASES OF LUNG ABSCESS—CONT'D

10	40	F	3 weeks after appendectomy	3 mo.	Left upper	10 cm. diam.	Postural drainage 3 months	Cured
11	44	M	Bronchiectasis	1 yr.	Right lower (developed empyema)	3.5 cm. diam.	Postural drainage 1 month	Died
12	43	M	Postpneumonic	2 mo.	Left upper	1 cm. diam.	Postural drainage 6 weeks	Cured
13	38	M	?	6 mo.	Right middle	2 cm. diam.	Postural drainage 6 weeks. Bronchoscopy	Died 1 year later
14	36	F	1 week after tonsillectomy	1 mo.	Left upper (central)	2.5 cm. long, 1 cm. diam.	Postural drainage 6 weeks	Cured
15	21	F	3 days after tonsillectomy	2 mo.	Right upper	3 cm. diam.	Too ill for specific treatment	Died in 3 weeks
16	42	M	Postpneumonic	1 mo.	Right upper	1.5 cm. diam.	Postural drainage 2 months	Cured
17	47	M	? Postpneumonic	2 mo.	Left upper	Large, multilocular	Postural drainage 3 weeks	Died
18	36	M	10 days after tooth extraction	1 yr.	Right upper	4 cm. diam.	Postural drainage 1 month	Improved
19	42	M	1 month after tooth extraction	4 mo.	Left upper	2.5 cm. diam.	Postural drainage 1 month	Cured
20	63	F	2 weeks after tonsillectomy	9 yr.	Left lower	2 cm. diam.	Postural drainage 3 months	Improved
21	31	M	Postpneumonic	1 mo.	Left upper (central)	1.5 cm. diam.	Postural drainage 1 month	Cured
22	24	F	3 days after tooth extraction	1 mo.	Right upper	1 × 2 cm.	Postural drainage 2 months	Cured

DATA IN TREATED CASES OF LUNG ABSCESS—CONT'D

CASE	AGE	SEX	ETIOLOGY	DURATION OF ABSCESS	LOBE INVOLVED	SIZE OF ABSCESS	TREATMENT	RESULT
23	31	F	2 weeks after tonsillectomy	2 mo.	Right upper	3 cm. diam.	Postural drainage 2 weeks	Died
24	43	M	3 weeks after hemorrhoidectomy. Postoperative pneumonia	1 mo.	Right upper	2 cm. diam.	Postural drainage 2 months	Cured
25	63	M	After cholecystectomy (embolism followed by abscess)	1 yr.	Right upper	4 cm. diam.	Not treated (in for diagnosis only)	Not followed
26	34	M	Postpneumonic	2 mo.	Left upper	8 cm. diam.	Postural drainage 5 months	Cured
27	35	M	Overcome by fumes of acetylene torches (? pneumonic)	2 mo.	Left lower	3.5 × 2 cm.	Postural drainage	Cured
28	24	F	3 weeks after tonsillectomy	2 mo.	Left upper	2.5 cm. diam.	Postural drainage 1 month	Died
29	45	M	3 weeks after tooth extraction	1 mo.	Right upper	1 cm. diam.	Postural drainage 6 weeks	Cured
30	31	M	After tonsillectomy	4 mo.	Right upper	2.5 × 3.5 cm.	Postural drainage 5 months	Unimproved. Lesions advanced. Patient lost
31	23	F	11 days after tonsillectomy	1 mo.	Right upper	1 cm. diam.	Postural drainage	Cured
32	33	M	Postpneumonic	1 mo.	Right upper	3 cm. diam.	Postural drainage 6 weeks	Improved

DATA IN TREATED CASES OF LUNG ABSCESS—CONT'D

33	32	F	1 week after hemorrhoidectomy	2 mo.	Right upper	2.5 cm. diam.	Postural drainage 5 weeks	Cured
34	21	F	8 days after tonsillectomy	1 mo.	Left upper	1.5 cm. diam.	Postural drainage 2 months	Cured
35	50	M	?	6 mo.	Left lower	5 cm. diam.	Postural drainage 2 weeks	Died
36	38	M	?	1 mo.	Right upper	1 cm. diam.	Postural drainage 2 weeks	Died 4 months later
37	26	F	10 days after tonsillectomy	1 mo.	Right upper	3.5 cm. diam.	Postural drainage 7 weeks	Cured
38	45	F	?	11 mo.	Right middle	3 cm. diam.	Postural drainage 1 week	Died of nephritis
39	24	F	4 days after tonsillectomy	1 mo.	Right upper, left upper	Rt.—2 cm. diam. Lt.—4 cm. diam.	Postural drainage 6 weeks	Improved
40	27	F	1 week after tonsillectomy	1 mo.	Left lower	5 cm. diam.	Postural drainage 5 weeks	Cured
41	24	M	1 week after tonsillectomy	3 mo.	Right lower	2 cm. diam.	Postural drainage 1 month	Cured
42	56	M	?	2 mo.	Left upper	1 cm. diam.	Not treated (in for diagnosis only)	Not followed
43	57	F	Followed influenza pneumonia	15 yr.	Right upper	3 cm. diam.	Not treated (in for diagnosis only)	Not followed
44	50	M	3 weeks after tooth extraction	1 mo.	Right lower	4 cm. diam.	1 stage drainage of abscess	Cured
45	55	M	Originated in bronchiectatic cavity	2 mo.	Right upper	4-5 cm. diam.	1 stage drainage of abscess	Died 1 week after operation

DATA IN TREATED CASES OF LUNG ABSCESS—CONT'D

CASE	AGE	SEX	ETIOLOGY	DURATION OF ABSCESS	LOBE INVOLVED	SIZE OF ABSCESS	TREATMENT	RESULT
46	40	M	5 weeks after appendectomy	1 mo.	Left lower	3-4 cm. diam.	1 stage drainage of abscess	Died 10 days after operation
47	30	M	Postpneumonic	10 mo.	Left lower	4 cm. diam.	2 stage drainage of abscess	Cured
48	45	M	?	1 mo.	Left lower (central)	2-3 cm. diam.	1 stage drainage of abscess	Died 3 days after operation
49	45	M	? Postpneumonic	3 mo.	Left upper (developed empyema)	6 cm. diam.	Drainage of abscess twice. Drainage of empyema	Died 1 day after third operation
50	41	M	10 days after tooth extraction	2 mo.	Right lower	2 cm. diam., 4 cm. long	Drainage of abscess 3 times	Improved
51	20	M	?	12 mo.	Left lower	2 cm. diam.	Operative drainage twice	Improved
52	49	M	8 days after inguinal herniorrhaphy (? postoperative pneumonia)	1 mo.	Left lower	3 cm. diam.	Operative drainage twice	Unimproved, died 4 months later
53	21	F	4 days after tonsillectomy	4 mo.	Right lower	8 cm. diam.	1 stage drainage of abscess	Cured
54	17	F	1 week after tonsillectomy	4 mo.	Left upper	3 cm. diam.	7 months medical treatment, then 1 stage drainage	Unimproved
55	59	F	Postpneumonic	2 mo.	Right lower	5 cm. diam.	1 stage drainage of abscess	Died of cerebral embolism
56	39	M	1 week after cholecystectomy	2 mo.	Right lower	4 cm. diam.	2 stage drainage of abscess	Cured

DATA IN TREATED CASES OF LUNG ABSCESS—CONT'D

57	36	M	?	3 mo.	Right upper (central)	2 cm. diam., 4 cm. long	2 stage drainage of abscess	Well 5 years, then recurrence and death
58	56	M	13 days after tooth extraction	1 mo.	Right lower	6.5 cm. diam.	1 stage drainage of abscess	Died 1 month after operation (bacteremia)
59	31	M	? Postpneumonic	11 mo.	Right upper	8 cm. diam.	1 stage drainage of abscess	Improved
60	17	M	3 days after tonsillectomy	1 mo.	Right middle	3.5 cm. diam.	1 stage drainage of abscess	Died 2 weeks after operation
61	19	F	11 days after tonsillectomy	1 mo.	Left upper	2.5 cm. diam., 3.5 cm. long	7 months medical treatment, then 2 stage drainage	Cured
62	50	M	? Postpneumonic	5 mo.	Right lower	1.5 cm. diam.	Operative drainage 3 times	Cured
63	26	M	? Postpneumonic	1 mo.	Right middle	2.5 cm. diam.	1 stage drainage of abscess	Cured
64	40	M	2 weeks after nasal operation	1 mo.	Right upper	8 cm. diam.	1 stage drainage of abscess	Died 2 weeks after operation
65	33	M	10 days after tonsillectomy	8 mo.	Left upper	Large multilocular	8 months pneumothorax, then 2 stage ant. and post. thoracoplasty	Died 2 weeks after operation
66	36	M	Postpneumonic	10 mo.	Right upper	2 cavities each 2.5 cm. and 5 cm. long	Attempted 1 stage drainage of abscesses	Died 2 weeks after operation
67	29	F	4 days after tonsillectomy	1 mo.	Left upper (developed empyema)	3 cm. diam.	Drainage of empyema: medical treatment for abscess	Cured

DATA IN TREATED CASES OF LUNG ABSCESS—CONT'D

CASE	AGE	SEX	ETIOLOGY	DURATION OF ABSCESS	LOBE INVOLVED	SIZE OF ABSCESS	TREATMENT	RESULT
68	59	M	3 days after cholecystectomy (? postpneumonic)	1 mo.	Left lower	7 cm. diam.	1 stage drainage of abscess	Died day of operation
69	41	F	2 weeks after teeth extraction	1 mo.	Left upper (developed empyema)	8 cm. diam., 14 cm. long	1 stage drainage of abscess. Drainage of empyema	Improved
70	47	M	1 week after teeth extraction	1 mo.	Right lower (central) (developed empyema)	2 cm. diam.	5 months medical treatment, then drainage of empyema	Died day of operation
71	40	F	?	4 yr.	Right upper	5 cm. diam.	Medical treatment 5 months, then drainage of abscess 5 times	Unimproved
72	24	M	? Postpneumonic	1 mo.	Right upper	3.5 cm. diam.	1 stage drainage of abscess	Cured
73	38	M	After gastric resection	3 mo.	Right lower (developed empyema)	3 cm. diam.	2 stage drainage of abscess. Drainage of empyema	Died of pulmonary embolism
74	41	M	? Postpneumonic	8 mo.	Left upper	4.5 cm. diam.	Medical treatment 9 months, then 2 stages of cautery lobectomy	Died 2 weeks after second stage
75	52	F	Postpneumonic	9 mo.	Right lower	6 cm. diam.	First stage of pneumectomy	Died 2 weeks after operation
76	49	M	Postpneumonic	18 mo.	Left lower	Multiple cavities averaging 1.5 cm. diam.	3 stages of cautery pneumectomy	Died of air embolism during 3rd operation

DATA IN TREATED CASES OF LUNG ABSCESS—CONT'D

77	19	M	?	2 mo.	Left lower (developed pyopneumothorax)	6 cm. diam.	Drainage of empyema	Died
78	41	M	After inguinal herniorrhaphy. (? postpneumonic)	10 mo.	Left lower	9 cm. diam.	Phrenicectomy, 4 stages of cautery lobectomy	Improved
79	44	F	2 days after teeth extraction	3 mo.	Right lower (developed empyema)	1.5 cm. diam.	Medical treatment 8 months, then 3 operations. Closed drainage of empyema. Partial thoracoplasty. Partial cautery lobectomy	Cured
80	18	F	?	4 mo.	Right lower (developed empyema)	5 cm. diam., 4.5 cm. long	Medical treatment of abscess, closed drainage of empyema	Cured
81	50	F	?	1 mo.	Right lower	Large multilocular	1 stage drainage of abscess	Improved
82	45	M	2 weeks after tonsillectomy	2 mo.	Right upper (developed pyopneumothorax)	2 cavities, 3 and 5 cm. diam.	Closed drainage of pyopneumothorax	Died 1 week after operation
83	36	M	Bronchiectasis (gassed in war)	yrs.	Right lower	Multiple cavities	Medical treatment for several years, then phrenicectomy and 11 stages of cautery lobectomy	Improved

DATA IN TREATED CASES OF LUNG ABSCESS—CONT'D

CASE	AGE	SEX	ETIOLOGY	DURATION OF ABSCESS	LOBE INVOLVED	SIZE OF ABSCESS	TREATMENT	RESULT
84	39	F	3 weeks after tooth extraction (novocaine)	9 mo.	Right middle	3 cm. diam.	9 months medical treatment, then 2 stage drainage of abscess	Cured
85	23	M	? Postpneumonic	1 mo.	Right lower (developed pyopneumothorax)	2 abscesses, 3 cm. diam., 2 cm. diam.	Drainage of pyopneumothorax	Cured
86	40	M	? Postpneumonic	8 mo.	Left upper	2.5 cm. diam.	7 months medical treatment, then 2 stage drainage of abscess	Cured
87	33	M	5 days after tonsillectomy	1 mo.	Left lower (developed pyopneumothorax)	2 abscesses, 2 cm. diam., 1.5 cm. diam.	Drainage of empyema	Died
88	25	M	6 days after gastrectomy	1 mo.	Right upper	6 cm. diam.	Medical treatment 2 months, then 1 stage drainage of abscess	Cured
89	30	M	?	1 mo.	Left lower (developed pyopneumothorax)	1 cm. diam.	Drainage of empyema	Died of brain abscess
90	43	M	1 week after teeth extraction	3 mo.	Right lower	3 cm. diam.	1 stage drainage of abscess	Died of brain abscess

REFERENCES

1. Bucher, C. J.: Abscess of the Lung: A Bacteriologic Study Based on One Hundred and Eighteen Cases, Am. J. M. Sc. **179**: 406, 1930.
2. Cherry, H. H.: The Rest Treatment of Non-tuberculous Pulmonary Abscess, Am. Rev. Tuberc. **25**: 634, 1932.
3. Cohen, J.: The Bacteriology of Abscess of the Lung and Methods for Its Study, Arch. Surg. **24**: 171, 1932.
4. Crowe, S. J., and Scarff, J. E.: Experimental Abscess of the Lung in the Dog, Arch. Surg. **16**: 176, 1928.
5. Cutler, E. C., and Hunt, A. M.: Postoperative Pulmonary Complications, Arch. Int. Med. **29**: 449, 1922.
6. Cutler, E. C., and Schlueter, S. A.: The Experimental Production of Abscess of the Lung, Ann. Surg. **84**: 256, 1926.
7. Cutler, E. C.: The Etiology of Postoperative Abscess of the Lung, Ohio State M. J. **24**: 109, 1928.
8. Cutler, E. C.: The Experimental Production of Abscess of the Lung, Am. J. Dis. Child. **38**: 683, 1929.
9. Dailey, L., and Daily, R. K.: Bronchoscopic and Esophagoscopic Observations in Tonsillectomies, Texas State J. Med. **23**: 277, 1927.
10. Dolley, F. S.: Internal Drainage of Lung Abscess by Extrapleural Compression, J. THORACIC SURG. **1**: 363, 1932.
11. Flick, J. B., Clerf, L. H., Funk, E. H., and Farrell, J. T.: Pulmonary Abscess: Analysis of One Hundred and Seventy-Two Cases, Arch. Surg. **192**: 1292, 1929.
12. Herrmann, L. G., and Cutler, E. C.: A Method for Experimental Production of Chronic Abscess of Lung, Proc. Soc. Exper. Biol. & Med. **26**: 28, 1928.
13. Heuer, G. J.: The Etiology and Treatment of Pulmonary Abscess, Surg. Gynec. Obst. **52**: 394, 1931.
14. Holloway, J. W., Schlueter, S. A., and Cutler, E. C.: The Relation of Immunity to the Experimental Production of Abscess of the Lung, Ann. Surg. **86**: 165, 1927.
15. Holman, Emile, Weidlein, I. F., and Schlueter, S. A.: A Method for the Experimental Production of Lung Abscess, Proc. Soc. Exper. Biol. & Med. **23**: 266, 1926.
16. Homans, J.: The Etiology and Clinical Features of Lung Abscess, Boston M. & S. J. **188**: 577, 1923.
17. Iglauer, S.: Aspiration of Blood Into Larynx and Trachea During Tonsillectomy Under Local Anesthesia: A Contribution to the Etiology of Lung Abscess, Ann. Otol. Rhin. & Laryng. **37**: 231, 1928.
18. King, D. S., and Lord, F. T.: Certain Aspects of Pulmonary Abscess From an Analysis of 210 Cases, Ann. Int. Med. **8**: 468, 1934.
19. Kline, B. S., and Berger, S. S.: Pulmonary Abscess and Pulmonary Gangrene —Clinical Course and Pathology, Arch. Surg. **18**: 481, 1929.
20. Kline, B. S., and Berger, S. S.: Pulmonary Abscess and Pulmonary Gangrene; an Analysis of Ninety Cases Observed in Ten Years, Arch. Int. Med. **56**: 753, 1935.
21. Lambert, A. V. P., and Weeks, C.: Experimental Production of Abscess of the Lung, Arch. Surg. **18**: 516, 1929.
22. Lilienthal, H.: Operative Treatment of Abscess of the Lung, Surg. Gynec. Obst. **53**: 788, 1931.
23. Longacre, J. J., and Herrmann, L. G.: Experimental Studies on Pulmonary Suppuration, Arch. Surg. **30**: 476, 1935.
24. Miller, J. A., and Lambert, A. V. P.: The Treatment of Abscess of the Lung, Am. J. M. Sc. **171**: 81, 1926.
25. Modern Medicine, Vol. I. Edited by Sir William Osler and Thomas McCrae, Philadelphia, 1913, Lea & Febiger.
26. Muller, George P.: Mortality and End-Results of Operations for Abscess of the Lung, Ann. Surg. **91**: 361, 1930.
27. Scarff, J. E.: The Experimental Production of Pulmonary Abscess: Etiologic Factors, Arch. Surg. **184**: 1960, 1929.
28. Schlueter, S. A., and Weidlein, I. F.: Postoperative Lung Abscess, Arch. Surg. **14**: 457, 1927.
29. Smith, D. T.: Experimental Aspiratory Abscess, Arch. Surg. **14**: 231, 1927.

30. Tuttle, W. M., and Cannon, P. R.: Effects of Local Immunization on Development of Experimental Abscesses of the Lung, Arch. Surg. **30**: 243, 1935.
31. Van Allen, C. M., Adams, W. E., and Hrdina, L. S.: Bronchogenic Contamination in Embolic Abscess of the Lungs, Arch. Surg. **192**: 1262, 1929.
32. Van Allen, C. M., Adams, W. E., and Hrdina, L. S.: Embolism in Bronchogenic Infection of the Lung, Arch. Surg. **192**: 1279, 1929.
33. Varney, P. L.: The Bacterial Flora of Treated and Untreated Abscesses of the Lung, Arch. Surg. **192**: 1002, 1929.
34. Wessler, H.: Lung Abscess and Bronchiectasis: A Clinical and Roentgenological Study of 100 Cases, Am. J. Roentgenol. **6**: 161, 1919.
35. Whittemore, W., and Balboni, G. M.: Non-tuberculous Bronchopulmonary Suppurative Lesions: Results of Treatment by Artificial Pneumothorax, Arch. Surg. **16**: 228, 1928.
36. Wood, C. B.: Pyopneumothorax Occurring as a Complication of Acute Pulmonary Suppuration, Am. J. Surg. **30**: 289, 1935.

TREATMENT OF LUNG ABSCESS

With Report of 100 Consecutive Cases*

Clyde I. Allen, M.D., and James F. Blackman, M.D.
Detroit, Mich.

THE continued high death rate from lung abscess makes it justifiable perhaps to present another series of cases, together with an analysis of previously reported series in an attempt to determine means of reducing this mortality rate.

Etiology.—The etiologic factors need be mentioned only briefly. As pointed out comparatively recently, the increasing number of surgical operations on the upper respiratory tract has increased the incidence of postoperative lung abscess. Whether this relationship is the result of aspiration or of embolic phenomena is not definitely settled. Experimentally there is much evidence to indicate that the production of abscesses by emboli may be the most likely method. Pneumonia and influenza may be predisposing or causative diseases. Foreign bodies and tumors, with resultant bronchial obstruction, will ever account for a fairly large number. Bacteremia and septicemia may be exciting causes.

Numerous as may be the etiologic agents, the fact remains that the lung is remarkably resistant to infection, and the production of abscesses is surprisingly infrequent.

Table I

Etiology of Lung Abscess

Group One—Following Operation on Respiratory Tract	
Tonsillectomies	18 cases
Dental extractions	3 cases
Submucus resections	2 cases
Plastic operation on nose	1 case
Total	24 cases

Local anesthesia 11 cases. HFH 6—Elsewhere 5.
General anesthesia 13 cases. HFH 2—Elsewhere 11.
Aver. 8 days. Min. 2 days. Max. 12 days.
Well—11(46%). Improved—4 (17%). Died—9 (37%).

In our own series of 100 cases, 24 followed operations on the upper respiratory tract. The details relating to this group are set forth in Table I. It is of interest to note that an almost equal number followed local and general anesthesia. There was almost an equal distribution

*From the Department of Surgery, Henry Ford Hospital.
Read before the Nineteenth Annual Meeting of the American Association for Thoracic Surgery at Rochester, Minn., May 4, 5, and 6, 1936.

in the right lower and left lower lobes. In the cases which followed tonsillectomies, three had postoperative hemorrhages sufficiently severe as to require treatment. Twelve cases, or 50 per cent of this group, had chest pain as the first symptom of pulmonary disease, and these were the cases in which the diagnosis was made from two to five days following operation. Those patients who had a longer interval between diagnosis of abscess and operation usually had a fever of unknown cause, until the onset of production of foul sputum.

An additional group of 10 cases followed a variety of operations as shown in Table II. In this group, all had general anesthetics. It is of some interest to compare the locations of the abscesses in the first and second operative groups. In the first group 29 per cent of the abscesses were in the upper or middle lobes; while in the second or miscellaneous group 50 per cent were in these locations. It might be argued that this offers some evidence in favor of the aspiration of infected material, rather than the occurrence of emboli as the exciting cause of the infection in the first group.

TABLE II

ETIOLOGY OF LUNG ABSCESS

Group Two—Following Other Operations	
Cholecystectomy	2 cases
Hernia repair	2 cases
Manipulation of spine	1 case
Amp. of arm	1 case
Resection of rectum	1 case
Closure of perforated peptic ulcer	1 case
Tubal pregnancy	1 case
Normal delivery	1 case
Total	10 cases (or 10%)

Local 0—General 10 (HFH 8—Elsewhere 2).
Aver. 9 days. Min. 3 days. Max. 17 days.
Infected cases 3. Clean cases 7.
Well—5. Died—4. Improved—lost track of—1.

Of the entire group of 100 cases, 18 were attributed to pneumonia, 8 of whom had been treated elsewhere and were seen by us only after the abscess was well developed. Of the 10 who were followed in our clinic from the onset of the illness, 7 showed some type of pneumococci in the sputum. In each instance the abscess was in the same lobe as the pneumonia. Of the 18 cases, 13 were well, 3 improved and considered on the way to complete recovery when last seen, and 2 died. The mortality rate in this group, therefore, is 11.1 per cent, which is a great deal lower than that for the postoperative cases shown in Tables I and II.

The etiologic factors in the remaining 48 cases are of a wide variety. In one case the abscess was diagnosed following a period of diabetic coma and may presumably be attributed to aspiration. One was the result of pressure of an aortic aneurysm, one occurred following whoop-

ing cough, and six were the result of obstruction by thoracic tumors. The remaining 38 cases were admitted to the hospital with the abscesses well developed, the preceding illnesses being given as head cold, sore throat, bronchopneumonia, influenza, or a chronic cough which gradually became productive. Careful questioning usually brought out evidence of some type of preceding illness which might have acted as the cause of the abscess.

Bacteriology.—Approximately 80 per cent of our group of 100 cases were caused by anaerobic and 20 per cent by aerobic organisms. In some instances the diagnosis of putrid lung abscess was made because of the presence of an extremely foul smelling sputum and the finding of fusiform bacilli or spirochetes or both in the slide, although cultures for anaerobic organisms were not made in all cases.

Symptomatology.—Little need be said here regarding the symptomatology of lung abscess, for its salient features are well known. It has been of interest to us to note the very great frequency in which pain over the site of the abscess was an early symptom. This fact may often be valuable in localizing an abscess. The varying fever, expectoration of pus laden sputum, extremely malodorous if caused by the anaerobic organisms, clubbing of the fingers, anorexia, loss of weight, sweating and cough are too well known to need elaboration.

Age and Sex.—Of the group of 100 patients, 81 were males and 19 were females. As to age incidence, they were divided into eight decades, 75 being in the third, fourth and fifth decades, the fourth having the greatest number with 34.

Diagnosis.—The diagnosis of lung abscess is frequently extremely difficult. At the same time, if proper treatment is to be instituted, accurate localization is imperative. A distinct fluid level in two views with the x-ray simplifies the matter, but so frequently this is lacking The expectoration of pus laden sputum, combined with a septic type of temperature and changes in the x-ray films indicative of an inflammatory condition, makes the diagnosis of abscess at least presumptive. The injection of lipiodol has not been especially valuable except as a means of differentiating bronchiectasis from lung abscess. X-ray films taken repeatedly and in oblique as well as the usual planes will often give the clue to exact localization. One factor not to be disregarded is the location of pain, which frequently is directly over the affected area. Occasionally, one still may see the recommendation of the use of the exploratory needle. It is our own feeling that this procedure is extremely dangerous. It has been recommended, also, that occasionally an exploratory thoracotomy should be done so that the surface of the lung may be palpated in order to locate exactly the inflammatory condition. This also seems to us to be an unjustifiable procedure.

One should never lose sight of the value of careful physical examination as a means of localizing a suspected abscess. All too often, however, the findings are equivocal; and one is left with the feeling, whether one likes to accept it or not, that the diagnosis must be made from the patient's history, state of health, the fact that pus laden sputum is being expectorated, and the x-ray findings.

Conservative Treatment.—Of the group of 100 patients, 55 were treated by conservative measures, which included phrenic nerve crushing, the use of pneumothoraces, postural drainage, the injection of neoarsphenamine and the ordinary supportive measures as used in all cases.

Postural drainage is, of course, of great value but should not be continued too long unless progressive improvement is occurring. It must be strictly supervised to be effective, and various positions must be utilized.

In our own series, neoarsphenamine injections were given at longer intervals and not continued so long as is recommended by some. The impression has, however, been gained that nothing more than a transient improvement resulted from this method of treatment.

Transfusions are of inestimable value in increasing resistance and have been used extensively, especially during the past ten years.

Table III shows the locations of the abscesses and the results obtained in those who have been treated conservatively. Of those who recovered and were well, the shortest time from the onset of the illness to recovery was 30 days, the longest 400 days, and the average 130 days. Of this same group, the shortest period of hospitalization was 7 days, the longest 90 days, and the average 44 days. Of the 14 patients who died, 3 had bronchiogenic carcinoma and died because of this rather than of the abscess itself. If these three are disregarded, the mortality rate in this group is, therefore, 21 per cent.

TABLE III

TREATMENT OF LUNG ABSCESS BY CONSERVATIVE METHODS

LOCATION	NO. OF CASES	WELL	IMPROVED	UNIMPROVED	DIED
RU	6	2	2		2
RM	11	6	2	1	2
RL	18	12	2		4
LU	7	1	3		3
LL	13	9		1	3
Total	55	30	9	2	14

For the entire group of 55 cases, the shortest elapsed time from the onset of the illness until the institution of treatment was 23 days, the longest 60 days, and the average 49 days.

Twelve of the 55 cases were treated with postural drainage and the ordinary supportive measures. Of these, 8 were well, 1 improved, 1 unimproved, and 2 died.

Ten of these patients were treated with supportive measures, postural drainage and bronchoscopic drainage. Of these, 7 were well, 1 improved and 2 died.

In addition to the therapeutic use of bronchoscopic drainage in these 10 cases, bronchoscopy was also utilized as a diagnostic procedure in a large percentage of the entire group. It had not been our feeling that it could be relied upon as the main therapeutic measure except in isolated instances. In a review of the literature, however, we were able to collect 650 cases in which bronchoscopic drainage was the principal treatment; and of these 398, or 61 per cent, were well, 20 per cent were improved, 12.5 per cent were unimproved, and 6 per cent had died. This is an exceptional record, and it perhaps behooves us to give this method of treatment more consideration than it has had in the past.

The evaluation of the use of phrenic nerve crushing in the treatment of lung abscess is difficult because it is invariably combined with other conservative measures, such as postural drainage and the administration of neoarsphenamine. It was used 26 times in our series. Of these patients, 12 were well, 3 improved, 10 unimproved and 1 died. This represents a high percentage of recovery but also a high percentage of unimprovement. Of those who were well following phrenic crushing, the elapsed time from diagnosis to operation varied from 5 to 47 days, the average being 25 days. The elapsed time in the remainder of the groups varied from 10 days to 2 years, the average time being 137 days. While this is undoubtedly an unfair comparison if taken exactly, because of the few whose operation was greatly delayed, the fact remains that the ones who did poorly had a longer interval before operation. This fact is easily explained on the basis of the formation of thick unyielding walls about the abscess. Of the 12 who recovered without further operative procedures, 7 were in the lower lobes. Of the 6 who died, 4 were in the right upper lobe, one in the right lower, and one in the left lower lobe.

After eliminating the 12 who were well and the 1 who died, of the remaining 13 cases, 9 eventually had open drainage, 1 closed drainage of a complicating empyema, and 3 were sufficiently improved when last seen to permit a reasonably accurate prognosis of complete recovery.

Of this group of 26 patients, 4 died, 1 following phrenic crushing and neoarsphenamine treatment who died of pneumonia, and 3 died following open drainage.

While it is true that the temporary paralysis of the diaphragm produced by phrenic nerve crushing undoubtedly decreases the ability of the patient to cough up the contents of the abscess, there are many instances in which this constitutes an excellent form of treatment and in properly selected cases may be expected to give a fairly high percentage of satisfactory results. Needless to say, it will be valueless unless satisfactory drainage into a bronchus is already established.

In our series of 100 cases, pneumothorax was attempted in 26. The shortest time that elapsed between the diagnosis of the abscess and the attempt at pneumothorax was 7 days and the longest time was 3 years. The average time for the groups was 107 days, which is hardly of any significance because of the few cases with the prolonged interval which greatly increased the average.

Of this group of 26 cases, satisfactory collapse was obtained in only 4 instances. The first of these had the collapse started within 30 days of the onset of illness and improved rapidly with complete recovery. One had the pneumothorax produced 90 days after the onset of symptoms and died of bronchiogenic carcinoma, the diagnosis of which was clouded by the pneumothorax. One patient had an injection three months after the onset of illness, showed marked improvement with cessation of expectoration of sputum and with a gain in weight, and then failed to return and could not be traced. The fourth one of this group had a pneumothorax started 240 days after the onset of his illness. He subsequently left our care, developed an empyema and died in another hospital.

A partial collapse was obtained in 17 of the 26 cases, and in each instance this was not over the location of the abscess. Four of the patients in this group recovered without further treatment even though the collapse was unsatisfactory. Four of these patients developed empyema, on the twentieth day in two instances and on the fortieth day in the other two. Two of these recovered with tube drainage and one with open drainage. One refused any type of treatment and could not be traced after leaving the hospital. Each of the four patients had an extremely stormy convalescence, and two had pressure pyopneumothoraces with the onset of the empyemas which necessitated the removal of air. Two improved sufficiently to become out-patients, ultimately failed to return for further observation or treatment and could not be traced. Six patients in the group were unimproved and subsequently had open drainage. Surprisingly enough, only one death occurred in this group. In this instance the pneumothorax was produced before good bronchial drainage had become established. The patient had a vomica of foul sputum, developed pneumonia on the opposite side five days later and died.

Five patients had such dense adhesions that a pneumothorax could not be produced.

Ten of the 26 patients on whom air injection was attempted subsequently had open drainage, 8 recovering and 2 dying.

It is of some interest to note that 3 patients were given pneumothoraces because of repeated hemoptyses, and all three continued to have severe hemoptyses following the production of the pneumothoraces. A detailed analysis of those patients on whom pneumothorax was attempted is shown in Tables IV and V.

As may be seen, 15 of the 26 patients were well, 3 were improved and sufficiently recovered to presuppose complete recovery, 5 were dead, 1 was untraced and presumably dead, and 2 improved but untraced.

TABLE IV

PNEUMOTHORAX TREATMENT IN LUNG ABSCESS. DIRECT RESULT IN 26 CASES

		WELL	IMPROVED	UNIMPROVED	DIED
Satisfactory collapse	4	1	1		2
Partial collapse	17	4	2	10	1
Unsuccessful	5			5	
		5	3	15	3

TABLE V

END-RESULTS OF PNEUMOTHORAX TREATMENT IN LUNG ABSCESS

Complete collapse	4	1 well 1 improved 1 died—bronchiogenic carcinoma 1 developed empyema—died elsewhere
Partial collapse	17	4 well 4 developed empyema: 2 tube dr.—well; 1 open dr.—well; 1 ref. treat.—(died—?) 2 improved—untraced 1 developed pneumonia (massive vomica)—died 6 open dr.: 4 well; 2 died
Unsuccessful	5	3 open dr.—well 2 improved

Of the 5 patients who developed empyema, 3 were well, one dead and one presumably dead. While the mortality rate is not greater than the group as a whole, the series is small and, considering the stormy course which the patients who develop empyema invariably go through and the fact that 50 per cent of these patients had to have further operative procedures, we believe that this method of treatment does not justify itself and should be abandoned.

Surgical Treatment.—Forty-five of the patients, excluding the 16 whose only surgical treatment was a phrenic nerve crushing, were treated surgically. Twenty-nine had drainage by the two-stage operation; 3 had only the first stage of the two-stage operation; 6 had open drainage of complicating empyema; 3 had closed drainage of complicating empyema; 3 had one-stage drainage and one had a thoracoplasty. The last was an abscess of six years' duration.

Of the 29 patients who had the two-stage operation, 14 were well, 3 improved and 12 died. Of the 12 who died, 3 had abscesses as the result of bronchial obstruction by carcinoma but were operated upon for the relief of symptoms. If these three cases are left out of consideration,

the mortality rate is 34.6 per cent. Another of these patients died of a massive hemorrhage from a peptic ulcer while making a satisfactory convalescence, so far as the abscess was concerned. Another died of an embolus two years after the second-stage operation. If it can be reasonably considered that these two deaths were not the result of the two-stage operation, the mortality rate for this group is 26.9 per cent.

There were 9 patients with abscesses who developed putrid empyemas. All were operated upon and one-third died. If this very serious complication arises, drainage must be established at once. In some cases the patient's condition may suddenly become so critical as to make rib resection inadvisable. With these patients, the introduction of an intercostal drainage tube may provide a temporary means of drainage until the condition improves, and occasionally complete recovery from both empyema and abscess will be the result. We have found it advantageous to utilize the Connell apparatus in providing constant suction in the tube in the chest, using cylinder oil in the container and allowing it to flow down the tube a drop at a time, thereby producing the constant suction.

There were three cases which appeared early in this series and many years ago whose abscesses were treated by one-stage drainage. All three died. We feel very strongly that this method of treatment should not be used.

Of the three patients who died following the first stage of the two-stage operation, one died on the day of operation, one on the third day and the other on the fifth day. The latter two had developed generalized empyema.

If open drainage is to be established, it is felt very strongly that it should not be unduly delayed and that it should invariably be done by the two-stage method. Whether compression is to be produced at the first stage without removal of the ribs is, I believe, a matter of choice. There are points in favor of this procedure. It has been our preference to remove the overlying ribs at the first stage, producing a fairly large window. The parietal pleura, if thickened, is very lightly seared with the actual cautery; and, after suturing the subcutaneous fascia deeply into the wound, a dichloramine T pack is placed in it. Otherwise, the pleura is painted with tincture of iodine and the pack inserted. This is left in place for a period of five to seven days. On its removal the two layers of pleura are firmly fixed together. A ventricular puncture needle is then used to locate definitely the abscess before proceeding to establish a wide funnel-shaped drainage tract down to it, burning very slowly with the actual cautery. The use of a suction tube in the wound has been found useful in carrying away all smoke and odor as the burning proceeds. One advantage of this method is that the ends of the ribs become well granulated over and protected before drainage is established.

In those cases which are caused by the anaerobic organisms we make no effort to close the resultant fistulas for a period of at least four or five months, after which time a closure can usually be effected by the use of a muscle flap transplant.

In those which develop a gangrene about the site of the original abscess with the formation of secondary abscesses, additional exposure with additional cauterization is necessary, carrying with it, thereby, the danger of hemorrhage. It is in this type of case that it is especially necessary that a wide exposure be made at the original operation so that cauterization of a sufficient area of lung tissue may be carried out.

Analysis of 34 Deaths.—An analysis of the deaths that occur in any series is probably of equal value to an analysis of the treatment of those who recover.

Six of the deaths were due to carcinoma which was complicated by abscess. In each of these cases the patient was seeking relief from the symptoms of the abscess rather than from any symptoms which might have been produced by the tumor. Two of the patients were diagnosed as tumor and abscess when first studied and were considered inoperable from the standpoint of the tumor and were therefore treated conservatively. One of these patients died in two months and one in four months. The third patient was treated with artificial pneumothorax, and the diagnosis of malignancy was thereby obscured until shortly before death. This patient lived eight months. Three of these patients were diagnosed as lung abscess and possible malignancy and were subjected to open drainage for the relief of the distressing symptoms of the abscess. One died shortly after operation because of a large abscess of one of the lower lobes caused by carcinoma of the esophagus. The two other patients were greatly relieved by the open drainage, one living twelve months and the other fourteen months following the operation. From the improvement in the condition of these two patients it would seem that no criticism could be made of draining an abscess caused by a carcinoma when the abscess is responsible in the main for the patient's symptoms. In those cases in which malignancy was suspected and not found in open drainage, obviously the biopsies were not taken from the area involving the tumor tissue. The inflammatory reaction in the lung was one of the causes of the difficulty in diagnosing the carcinoma. The one case in this group of six which can be very definitely criticized is the one in which an artificial pneumothorax was produced before the diagnosis was completed. As it turned out, this was the only patient in the group who might have been a candidate for radical surgery.

In the group which was treated by surgical measures, there are four cases which we feel are open to criticism. Two of these had been treated with pneumothoraces and at operation the lung was still partially collapsed. In one of these two cases, an attempt was nevertheless made to suture the parietal and visceral pleura and the patient died very

suddenly after returning to her room, the cause of death being obscure but possibly being pleural shock. In the second of these two cases, the abscess was aspirated through nonadherent pleura, and the patient died four days later of empyema. The possibility of repetition of these occurrences has long ago been eliminated by changes in operative methods. In the third case, at operation an empyema was encountered with direct communication with an abscess cavity, and tube drainage was employed instead of leaving the wound wide open. Although the wound was opened widely several days later, the patient went on to death, and it is possible that the delay in providing more adequate drainage was a contributing factor. The fourth case had intercostal tube drainage established for an empyema complicating an abscess, and this was continued for ten days before open drainage was provided. In this instance the patient's general condition was not good, and an attempt was being made to provide drainage by the more conservative method, and this probably was a mistake.

In the group of cases which were treated by conservative methods, we feel that there were three which may be criticized. In the first, the abscess was caused by stenosis of the bronchus due to an aortic aneurysm which was treated by repeated bronchoscopies, and this patient died of a rupture of the aneurysm. The second case was treated by artificial pneumothorax before adequate bronchial drainage had been established, and, when the abscess ruptured into the bronchus, there was such profuse drainage that the patient almost died. He developed a pneumonia and did die five days later. The third patient was treated by pneumothorax, was allowed to go home before his treatment was completed, and developed an empyema and died as a result of this.

There were six patients who died following open drainage as the result of the spread of the inflammatory process. In these cases there developed multiple areas of gangrene with subsequent abscess formation which in spite of repeated drainages resulted in gradual loss of strength and death. In these cases, criticism probably is that a wider area of exposure should have been made originally so that a wider area of destruction of involved tissue could have been carried out.

There were three patients who died of postoperative hemorrhage: one at operation, one five days after operation, and one twelve days after operation. There were seven patients who had a very rapidly spreading process which could not be controlled long enough to prepare the patient properly for operation. Two of these died, one on the seventh and one on the twentieth day following the operation.

There were five patients who had complications severe enough to cause death. One patient died of a metastatic brain abscess, two of embolism, one of osteomyelitis of the cervical vertebra which followed a tonsillectomy, and one patient died from a massive hemorrhage from a duodenal ulcer following a smooth postoperative course, after the drain-

age of the lung abscess. One of the patients who died of embolism had been cured of his abscess two years previously and died following an attempt to close a bronchial fistula.

TABLE VI

ETIOLOGY, LOCATION, AND RESULT IN 100 CASES

		NUMBER	WELL	IMPROVED	UNIMPROVED	DIED	MORTALITY
Operations on the nose, mouth and throat	RU	3		1		2	66.6%
	RM	4	2	1		1	25.0%
	RL	7	4	2		1	14.1%
	LU	1				1	100.0%
	LL	8	4			4	50.0%
Total:		23	10	4		9	37.5%
Operations on other parts of the body than those above	RU	2	1			1	50.0%
	RM	1	1				0.0%
	RL	2	1			1	50.0%
	LU	2	1	1			0.0%
	LL	3	1			2	66.6%
Total:		10	5	1		4	40.0%
Pneumonia	RU	2	1			1	50.0%
	RM	6	4	2			0.0%
	RL	3	2	1			0.0%
	LU	2	1			1	50.0%
	LL	5	5				0.0%
Total:		18	13	3		2	11.9%
Uncertain: head cold, influenza, cough, etc.	RU	7	2	2		3	42.8%
	RM	7	4		1	2	28.6%
	RL	12	7	1		4	33.3%
	LU	7	4	2		1	14.2%
	LL	5	3		1	1	20.0%
Total:		38	20	5	2	11	28.8%
Carcinoma of bronchus 5 Carcinoma of esophagus 1	RU RM						
	RL	3				3	100.0%
	LU	1				1	100.0%
	LL						
Total:		6				6	100.0%
2 foreign body (fb) 1 aortic aneurysm (aa) 1 diabetic coma (dc) 1 whooping cough (wc)	RU RM RL LU	3	fb	wc		fb	33.3%
	LL	2	dc			aa	50.00%
Total:		5	2	1		2	40.0%
Total for 100 cases		100	50	14	2	34	34.0%
By the elimination of six patients who died of carcinoma:							
Total for 94 cases		94	50	14	2	28	29.7%

Case Reports Collected From the Literature.—From among the articles which are listed in the bibliography, we were able to collect a series of 2,114 case reports of the treatment of lung abscess. Of these, 1,086,

or 51.4 per cent, were treated conservatively and 1,028, or 48.6 per cent, were treated by radical surgical procedures. It was interesting to note that the mortality rate in these two groups of cases was almost exactly the same, 34.4 per cent in the group treated conservatively and 34.2 per cent in the group which was treated surgically.

Of the group which was treated by surgical measures, it was not possible to make a detailed analysis from the information available except in a comparatively small number of the cases. However, of those who had surgical treatment 43.4 per cent were well, 19 per cent were improved, 3.2 per cent were unimproved, and 34.2 per cent died.

In that group whose main treatment consisted of the ordinary supportive measures together with arsenical preparations, 68 were reported, of whom 47 per cent were well, 33 per cent improved, 10 per cent unimproved and 8.8 per cent died. While this is, comparatively, a small number from so large a group of cases, the results are sufficiently good to warrant a more thorough consideration of this type of treatment. In the hands of most of those who have written, this type of treatment has not been of great value.

From this series of collected cases, 144 were treated by pneumothoraces together with supportive measures: 46 per cent were reported as well, 11.8 per cent as improved, 25 per cent as unimproved and 16.6 per cent as dead. Of this group of 144 patients, only 9, or 6.2 per cent, are reported as having developed empyemas and all of these died.

Another group of 107 cases had as the principal treatment postural drainage. Of these 43 per cent were well, 18.7 per cent were improved, 12.1 per cent were unimproved and 26 per cent died.

A report of those patients who had bronchoscopic drainage as the principle form of treatment has been given earlier in this paper.

If one were forced to draw conclusions from these figures which have been reported, they would be that the forms of treatment which offer the highest percentage of recoveries and the lowest percentage of deaths are the treatments with pneumothoraces, arsenicals, postural drainage and bronchoscopic drainage. It must be borne in mind, however, that those who go on to a more radical type of treatment are the failures which occur with conservative treatment. The only logical conclusion therefore is that the treatment, if it be conservative, must be allowed to remain so only so long as the patient is becoming progressively better, and that the delay in instituting radical treatment must not be unduly prolonged.

SUMMARY

In our own series of 100 cases the mortality rate was 34 per cent. For the first 50 cases it was 42 per cent, and for the last 50 cases, all of whom were treated during the last five years, it was 26 per cent. Of the 34 patients who died, however, 6 died of carcinomas which were pro-

ducing abscesses by obstruction. Of the remaining 94 cases therefore the mortality rate was 29.7 per cent. This reduced mortality is the result of the things we have learned in the treatment of the earlier cases and from the experience of others. It may well be attributed to earlier diagnosis and institution of treatment, closer cooperation between internist and surgeon, more accurate localization and the institution of more radical treatment earlier when conservative treatment has failed. With continued attention to this problem, further reduction in mortality rate may be anticipated.

From the technical standpoint, the following conclusions may be drawn:

1. Conservative treatment should not be continued unless progressive improvement is being accomplished, regardless of the time element.

2. Phrenic nerve crushing is sufficiently valuable to make its use advisable in certain selected cases.

3. The treatment with arsenicals, in the hands of some, has given good results and perhaps deserves a wider trial than it has had.

4. Treatment by bronchoscopic drainage has not proved of great value in most hands except as a diagnostic procedure. Its greatest value, probably, is for those abscesses which are caused by foreign bodies.

5. It seems apparent that the use of pneumothorax in lung abscess treatment is not justifiable.

6. Operative drainage should always be done in two stages.

7. A wide area for drainage should be established.

8. The drainage tract down to the abscess should be made with the actual cautery.

9. Tube drainage of complicating empyema is sometimes of value where the patient's condition is so critical as to make a more radical operation too hazardous.

REFERENCES

(1) Adams, W. E.: Rapid Healing of Embolic Abscess in Atelectatic Lobes, Proc. Soc. Exper. Biol. & Med. **29**: 539, 1932. (2) Adams, W. E., and Vorwald, A. J.: Experimental Production of Bronchiogenic Abscess of Lung, Proc. Soc. Exper. Biol. & Med. **30**: 463, 1933. (3) Alexander, J., and Buckingham, W. W.: Treatment of Non-Tuberculous Suppurative Pneumonitis, Abscess and Bronchiectasis, J. A. M. A. **95**: 1478, 1930. (4) Alexander, J., and Haight, C.: Abscess of Lung, J. Michigan M. Soc. **32**: 637, 1933. (5) Allen, D. S.: Etiology of Abscess of Lung; Experimental and Clinical Studies, Arch. Surg **16**: 179, 1928. (6) Archibald, E., and Brown, A. L.: Action of Cough Upon Material in Tracheo-Bronchial Tract; Experimental Study, Am. Rev. Tuberc. **16**: 111, 1927. (7) Archibald, E., and Brown, A. L.: Cough; Its Action on Material in Tracheo-Bronchial Tract; Experimental Study, Arch. Surg. **16**: 322, 1928. (8) Aschner, P. W.: Pathology of Lung Suppuration, Ann. Surg. **75**: 321, 1922. (9) Ascoli, M., and Boniades, A.: Experimental Production of Inflammatory and Suppurative Conditions of Lung, Arch. Surg. **25**: 1074, 1932. (10) Balboni, G. M.: Artificial Pneumothorax Discussion, Boston M. & S. J. **171**: 422, 1914. (11) Ballon, D. H., and Ballon, H. C.: Value of Bronchoscopic Injection of Lipiodol in Diagnosis and Treatment of Bronchiectasis, Lung Abscess and Tuberculosis, J. Laryng. & Otol. **44**: 153, 1929. (12) Ballon, H. C.: Value of Lipiodol in Diagnosis and Treatment of Abscess of Lung, Surg. Gynec. Obst. **44**: 1, 1927. (13) Belk, W. P.: Pulmonary Infections by Friedländer's Bacillus, J. Infect. Dis. **38**: 115, 1926. (14) Bendove, R. A.: Selective Therapeutic Pneumothorax in Acute Pulmonary Suppuration, Am. J. Surg. **19**: 49, 1933. (15) Berington Ward, L. E.: Pneumococcal Abscess of the Lung in Children, Lancet **1**: 1090, 1913. (16) Berry, F. D.: Massive Atelectasis Complicating Paravertebral Thoracoplasty for Pulmonary Tuberculosis, Arch. Surg. **18**: 487, 1929. (17) Bethune, N.: Note on Bacteriological Diag. of Spirochaetosis of Lungs, Canad. M. A. J. **20**: 365, 1929. (18) Bevan, A. D.: Abscess of the Lung—An Important Sequel to Tonsillectomy, Surg. Clin. Chicago **2**: 921, 1918. (19) Brunn, H., and Faulkner, W. B., Jr.: Intra-Bronchial Drainage; Its Importance in Diagnosis

and Treatment of Pulmonary Suppurations, Surg. Gynec. Obst. 51: 115, 1930. (20) Brunn, H.: Lung Abscess, J. A. M. A. 103: 1999, 1934. (21) Bucher, C. J.: Abscess of Lung; Bacteriologic Study Based on 118 Cases, Am. J. M. Sc. 179: 406, 1930. (22) Bullowa, J. G. M.: Pulmonary Abscess Following Aspiration of Sea Water, M. Rec. 95: 302, 1919. (23) Burel, L. S. T.: Discussion on Diagnosis and Treatment of Abscess of the Lung, Proc. Roy. Soc. Med. 25: 2: 1149, 1932. (24) Bushnell, J. E.: Subpleural Pulmonary Abscess, Am. J. M. Sc. 112: 419, 1896. (25) Cameron, H. C.: A Note on So-Called Metastatic Pulmonary Cerebral Abscess, Guys Hosp. Rep. 61: 69, 1907. (26) Carmody, T. E.: Treatment of Suppurative Conditions of Lung, New York M. J. 115: 742, 1922. (27) Carmody, T. E.: Pulmonary Abscess Following Tonsillectomy; Laryngologic Aspect, Arch. Otolaryng. 11: 200, 1930. (28) Castellani, A.: Note on a Peculiar Form of Haemoptysis With Presence of Numerous Spirochaetae in the Expectoration, Lancet 1: 1384, 1906. (29) Cecil, R. L., and Blake, F. G.: Studies in Experimental Pneumonia; Production of Pneumococcus Lobar Pneumonia in Monkeys, J. Exper. Med. 31: 403, 1920. (30) Chandler, F. G. & Others: Discussion on Treatment of Lung Abscess, Proc. Roy. Soc. Med. 23: 757, 1930. (31) Chandler, F. G.: Treatment of Lung Abscess, Clin. J. 59: 541, 1930. (32) Cherry, H. H.: Rest Treatment of Non-Tuberculous Pulmonary Abscess, Am. Rev. Tuberc. 25: 634, 1932. (33) Clarke, T. W., and Marine, D.: Pulmonary Gangrene Following Foreign Bodies in the Bronchi, Am. J. M. Sc. 131: 505, 1906. (34) Clendening, L.: Cause of Lung Abscess After Tonsillectomy, J. A. M. A. 74: 941, 1930. (35) Clendening, L.: Abscess of Lung, Laryngoscope 32: 128, 1922. (36) Clerf, L. H.: Pulmonary Abscess Following Tonsillectomy; Bronchoscopic Considerations, Arch. Otolaryng. 11: 192, 1930. (37) Clerf, L. H.: Bronchoscopic Treatment of Bronchiectasis and Lung Abscess, Am. Rev. Tuberc. 24: 605, 1931. (38) Clerf, L. H.: Pulmonary Abscess Following Tonsillectomy; Bronchoscopic Considerations, Tr. Am. Bronch. Soc. 12: 38, 1929. (39) Clerf, L. H.: Pulmonary Abscess, Bronchiectasis and Pulmonary Neoplasms; Bronchoscopic Aspects, Radiology 20: 365, 1933. (40) Cleveland, M.: Postoperative Pneumonitis, Surg. Gynec. Obst. 28: 282, 1919. (41) Coakley, C. G.: Lung Abscess, Laryngoscope 26: 1008, 1916. (42) Cohen, J.: Bacteriology of Abscess of Lung and Methods for Its Study, Arch. Surg. 24: 171, 1932. (43) Cohen, J.: Anaerobic Gram-Negative Bacilli Isolated From Abscess of Lung; Toxin Production as Demonstrated by Schwartzman Phenomenon, J. Infect. Dis. 52: 185, 1933. (44) Colard, A., and Caspers, F.: A Propos des Injections Intraveineuses d'Alcool dans les Suppurations Pulmonaires Non-Tuberculeuses, Scalpel 86: 1349, 1933. (45) Collier, E.: Pulmonary Gangrene Following Lingual Carcinoma, Brit. M. J. 2: 906, 1930. (46) Connors, J. F.: Treatment of Lung Abscess and Empyaema by Packing, Ann. Surg. 94: 38, 1931. (47) Corper, H. J.: Pulmonary Aspiration of Particulate Matter, Normally and During Anesthesia, J. A. M. A. 78: 1858, 1922. (48) Crowe, S. J., and Scarff, J. E.: Experimental Abscess of Lung in Dog, Arch. Surg. 16: 176, 1928. (49) Cummings, G. O.: Treatment and Management of Non-Tuberculous Pulmonary Abscess, With Special Reference to Series of 25 Consecutive Cases, Arch. Otolaryng. 19: 684, 1934. (50) Cutler, E. C., and Hunt, A. M.: Postoperative Pulmonary Complications, Arch. Surg. 1: 114, 1920. (51) Cutler, E. C., and Hunt, A. M.: Postoperative Pulmonary Complications, Arch. Int. Med. 29: 449, 1922. (52) Cutler, E. C., and Schlueter, S. A.: Experimental Production of Abscess of Lung, Ann. Surg. 84: 256, 1926. (53) Dick, G. F.: Fusiform Bacilli Associated With Various Pathological Processes, J. Infect. Dis. 12: 191, 1913. (54) Dunham, E. K.: Infection in Mediastinum in Fulminating Empyema, Surg. Gynec. Obst. 35: 288, 1922. (55) Edwards, A. T., & Others: Discussion on Intrathoracic Tumors, Proc. Roy. Soc. Med. 23: 760, 1930. (56) Eggers, C.: Lung Abscess Complicated and Hidden by Empyema, Arch. Surg. 12: 338, 1926. (57) Eggers, C.: Treatment of Suppuration of Lung, Ann. Surg. 87: 485, 1928. (58) Eggers, C., and Kernan, G.: Acute Pulmonary Suppuration; Selective Action of Artificial Pneumothorax in Treatment of This Disease, Arch. Surg. 16: 279, 1928. (59) Eisentdrath, D. N.: Surgery of Pulmonary Abscess Gangrene of Bronchiectasis Following Pneumonia, Philadelphia M. J. 8: 786, 1901. (60) Elkin, D. C.: Subphrenic Abscess, Internat. Clin. 1: 147, 1931. (61) Ermatinger, L. H.: Microorganisms of Lung Abscess and Bronchiectasis, J. Infect. Dis. 43: 391, 1928. (62) Faulkner, W. B., Jr.: Internal Drainage; Its Application in Pulmonary Suppuration, J. A. M. A. 95: 1325, 1930. (63) Faulkner, W. B., Jr.: Diagnostic Pneumothorax, Guide in Localization, Treatment and Prognosis of Pulmonary Abscess, Am. J. Surg. 14: 646, 1931. (64) Fetterolf, G., and Fox, H.: Reaction of Para-Tonsillar Tissues to Tonsillectomy; Study in Etiology of Post-Tonsillectomy Pulmonary Abscess, Am. J. M. Sc. 166: 802, 1923. (65) Fischer, L., and Cohen, A. J.: Pulmonary Abscess in Adults Following Tonsillectomy Under General Anesthesia, With Report of Cases, J. A. M. A. 77: 1313, 1921. (66) Fischer, A. E.: Pulmonary Sequelae of Tonsillectomy in Children, Am. J. M. Sc. 176: 253, 1928. (67) Flack, R. A.: Pyothorax From Fusospirochaetal Infection, Arch. Int. Med. 56: 790, 1935. (68) Flick, J. B.: Lung Abscess, Ann. Surg. 84: 323, 1926. (69) Flick, J. B., Clerf, L. H., Funk, E. H., and Farrel, J. T., Jr.: Pulmonary Abscess, Analysis of 172 Cases, Arch. Surg. 19: 1292, 1929. (70) Flick, J. B.: Pulmonary Abscess, Bronchiectasis and Pulmonary Neoplasms; Surgical Aspects, Radiology 20: 367, 1933. (71) Forbes, K. H.: Lung Abscess Following Inhalation of Foreign Body, Laryngoscope 28: 197, 1918, (72) Frank, I.: Lung Abscess as Sequel to Tonsillectomy, Laryngoscope 27: 474, 1917. (73) Frank, I.: Pulmonary Abscess Following Tonsillectomy, Ann. Otol. Rhin. & Laryng. 41: 550, 1932. (74) Frank, L. W.: Pulmonary Abscess, Ann. Surg. 95: 675, 1932. (75) Funk, E. H.: Pulmonary Abscesses, Bronchiectasis and Pulmonary Neoplasms; Clinical Aspects, Radiology 20: 353, 1933. (76) Georg, C.: Abscess of the Lung Following Fracture of the Ribs, Arch. Surg. 18: 531, 1929. (77) Gatewood, W. L.: Tonsillectomy; Local vs. General Anesthesia in Relation to Lung Abscess, Laryngoscope 39: 171, 1929. (78) Glowacki, B. F.: Pulmonary Abscess, Laryngoscope 33: 153, 1923. (79) Goldberg, B., and Bisenthal, M.: Treatment of Acute Living Abscess by Artificial Pneumo-Thorax, Am. Rev. Tuberc. 3: 169, 1919. (80) Graham, E. A.: Surgical Treatment of Pulmonary Suppuration in Children, J. A. M. A. 87: 806, 1926. (81) Graham, E. A.: Treatment of Pulmonary Suppuration, Ann. Surg. 86: 174, 1927. (82) Green, N. W.: Positive-Pressure Methods of Artificial Respiration, Surg. Gynec. Obst. 2: 512, 1906. (83) Greer, A. E.: Empyema, J. A. M. A. 66: 1019, 1916. (84) Greer, A. E.: Lung Abscess, Am. J. M. Sc. 169: 345, 1925.

(85) Halpin, J. A., Ellis, J. W., and Puckett, H. L.: Pulmonary Abscess, Am. J. Surg. **23**: 547, 1934. (86) Harkavy, J.: Pathogenesis of Aspiratory Abscess of Lung; Its Possible Relationship to Abscess of Lung Following Tonsillectomy, Arch. Int. Med. **43**: 767, 1929. (87) Harrington, S. W.: Amebic Hepatic, Subphrenic and Pulmonary Abscesses, Arch. Surg. **21**: 1146, 1930. (88) Harrington, S. W.: Surgical Treatment of Bronchiectasis and Lung Abscess, Am. Rev. Tuberc. **24**: 612, 1931. (89) Hartwell, J. A.: Abscess of Lung, Ann. Surg. **72**: 333, 1920. (90) Hedblomm, C. A.: Pulmonary Suppuration, M. Rec. **96**: 441, 1919. (91) Hedblomm, C. A.: Graded Extrapleural Thoracoplasty in Treatment of Diffuse Unilateral Bronchiectasis, Arch. Surg. **8**: 394, 1924. (92) Hedblomm, C. A.: Surgical Treatment of Acute Pulmonary Abscess and Chronic Pulmonary Suppuration With Especial Reference to Post-Tonsillectomy Type, J. A. M. A. **83**: 1577, 1924. (93) Hedblomm, C. A., Joannides, M., and Rosenthal, S.: Pulmonary Abscess Experimental Studies, Ann. Surg. **88**: 823, 1928. (94) Hedblomm, C. A., and Van Hazel, W.: Acute Post-Tonsillectomy Pulmonary Abscess of Right Upper Lobe; Complete Cure in 8 Weeks Following 2 Stage Thoracotomy Drainage Operation, S. Clin. North America **12**: 1145, 1932. (95) Hedblomm, C. A.: Treatment of Pulmonary Abscess, West J. Surg. **41**: 125, 1933. (96) Herb, I. C.: Post-Operative Lung Complications, J. A. M. A. **79**: 339, 1922. (97) Herrmann, L. G., and Cutler, E. C.: Experimental Production of Chronic Abscess of the Lung, J. Clin. Investigation **7**: 501, 1929. (98) Heurer, G. J.: Etiology and Treatment of Pulmonary Abscess, Surg. Gynec. Obst. **52**: 394, 1931. (99) Heurer, D. J., and MacCready, P. M.: Lung Abscess, Arch. Surg. **6**: 337, 1923. (100) Hitzrot, J. M.: Lobectomy for Bronchiectasis, Ann. Surg. **71**: 785, 1920. (101) Holman, E.: Etiology of Post-Operative Pulmonary Abscess, Ann. Surg. **83**: 240, 1926. (102) Holman, E., Weidlein, I. F., and Schlueter, S. A.: Method for the Experimental Production of Lung Abscess, Proc. Soc. Exper. Biol. & Med. **23**: 266, 1926. (103) Holman, E., Chandler, L. R., and Cooley, C. L.: Experimental Studies in Pulmonary Suppuration, Surg. Gynec. Obst. **44**: 328, 1927. (104) Holman, E.: Post-Tonsillectomic Pulmonary Abscess; Factors in Healing, Arch. Otolaryng. **11**: 287, 1930. (105) Holman, E.: Fundamental Principles Underlying Treatment of Intrapulmonary Abscesses and Persistent Bronchial Fistulae, West. J. Surg. **38**: 489, 1930. (106) Holloway, J. W., Schlueter, S. A., and Cutler, E. C.: Relation of Immunity to Experimental Production of Abscess of Lung, Ann. Surg. **86**: 165, 1927. (107) Homans, J.: Etiology and Clinical Features of Lung Abscess, Boston M. & S. J. **188**: 577, 1923. (108) Hunter, J. D.: Abscess of the Lung, Proc. Roy. Soc. Med. **25**: 1135, 1932. (109) Iglauer, S.: Aspiration of Blood Into Larynx and Trachea During Tonsillectomy Under Local Anesthesia; Contribution to Etiology of Lung Abscess, Ann. Otol. Rhin. & Laryng. **37**: 231, 1928. (110) Jackson, C.: Chronic Nonspecific Infections of Lungs; Their Bronchoscopic and Esophageal Phases, J. A. M. A. **87**: 729, 1926. (111) Jackson, C.: Suppurative Diseases of Lung Due to Inspirated Foreign Body Contrasted With Those of Other Etiology, Surg. Gynec. Obst. **42**: 305, 1926. (112) Jackson, C.: Suppurative Diseases of Lung, Brit. M. J. **2**: 702, 1930. (113) Jackson, C.: Suppurative Diseases of Lung; Report on Series of Bronchoscopic Observations, J. Laryng. & Otol. **46**: 73, 1931. (114) Jackson, C., and Jackson, C. L.: Postoperative Pulmonary Complications, Internat. Clin. **4**: 151, 1932. (115) Joannides, M.: Etiology of Pulmonary Abscess, Surg. Gynec. Obst. **47**: 449, 1928. (116) Jones, F. S.: An Organism Resembling Bacillus Actinoides Isolated From Pneumonic Lungs of White Rats, J. Exper. Med. **35**: 361, 1922. (117) Jones, H. W.: Thoracoplasty for Empyema With Bronchial Fistula and for Chronic Lung Abscess, Mil. Surgeon **66**: 791, 1930. (118) Keefer, C. S.: Pleural and Pulmonary Complications of Aortic Aneurysm, Ann. Int. Med. **8**: 72, 1934. (119) Keiper, G. F.: Tonsil Question Up to Date, Laryngoscope **31**: 777, 1921. (120) Keller, W. L.: Complications Incident to Inaccurate or Delayed Diagnosis in Suppuration of Lungs and Pleura, Bull. N. Y. Acad. Med. **7**: 202, 1931. (121) Kelly, R. E.: Intratracheal Insufflation of Ether, Brit. M. J. **2**: 617, 1912. (122) Kernan, J. D.: Abscess of Lung Relieved by Bronchoscopy; Report of Cases, Arch. Surg. **16**: 215, 1928. (123) King, D. S., and Lord, F. T.: Certain Aspects of Pulmonary Abscess From Analysis of 210 Cases, Ann. Int. Med. **8**: 468, 1934. (124) Kline, B. S., and Berger, S. S.: Pulmonary Abscess and Pulmonary Gangrene; Analysis of 90 Cases Observed in Ten Years, Arch. Int. Med. **56**: 753, 1935. (125) Kline, B. S., and Winternitz, M. C.: Experimental Production of Pneumonia, J. Exper. Med. **21**: 304, 1915. (126) Kline, B. S.: Experimental Gangrene, J. Infect. Dis. **32**: 481, 1923. (127) Kline, B. S., and Berger, S. S.: Spirochetal Pulmonary Gangrene Treated With Arsphenamine, J. A. M. A. **85**: 1452, 1925. (128) Kline, B. S.: Pulmonary Abscess and Pulmonary Gangrene, J. A. M. A. **90**: 2008, 1928. (129) Kline, B. S., and Berger, S. S.: Pulmonary Abscess and Pulmonary Gangrene; Clinical Course and Pathology, Arch. Surg. **18**: 481, 1929. (130) Kline, B. S.: Pathology of Lung Bronchiectasis and Lung Abscess, Am. Rev. Tuberc. **24**: 626, 1931. (131) Kornblum, K.: Roentgen-Ray Diagnosis of Pulmonary Infections With Friedländer Bacillus, Am. J. Roentgenol. **19**: 513, 1928. (132) Kramer, R.: Bronchoscopy in Pulmonary Suppuration, S. Clin. North America **8**: 377, 1928. (133) Lamar, R. V., and Meltzer, S. J.: Experimental Pneumonia by Intrabronchial Insufflation, J. Exper. Med. **15**: 133, 1912. (134) Lambert, A. V. S., and Miller, J. A.: Abscess of Lung, Arch. Surg. **8**: 446, 1924. (135) Lambert, A. V. S.: Pulmonary Abscess and Its Treatment From Surgical Standpoint, N. Y. State J. Med. **27**: 47, 1927. (136) Lambert, A. V. S., and Weeks, C.: Experimental Production of Abscess of the Lung, Arch. Surg. **18**: 516, 1929. (137) Lehmann, J. E.: Pulmonary Abscess, Canad, M. A. J. **10**: 1090, 1920. (138) Lemon, W. S.: Abscess of Lung, Canad. M. A. J. **10**: 1079, 1920. (139) Lemon, W. S.: Aspiration, Arch. Surg. **12**: 187, 1926. (140) Lemon, W. S., Vinson, P. P., Moeroch, H. J., and Wood, H. G.: Clinic on Abscess of Lung, M. Clin. North America **18**: 429, 1934. (141) Lewis, D.: Lung Abscess Following Tonsillectomy, Internat. Clin. **2**: 84, 1918. (142) Lilienthal, H.: Abscess of the Lung, Incision and Drainage. Thoracotomy, Ann. Surg. **59**: 855, 1914. (143) Lilienthal, H.: Resection of Lung for Suppurative Infections, Ann. Surg. **75**: 257, 1922. (144) Lilienthal, H.: Non-Tuberculous Pulmonary Suppuration: Comparison of Operations and Their Results, Arch. Surg. **16**: 206, 1928. (145) Lilienthal, H.: Operative Treatment of Abscess of the Lung, Surg. Gynec. Obst. **53**: 788, 1931. (146) Lockwood, A. L.: Abscess of Lung, Surg. Gynec. Obst. **35**: 461, 1922. (147) Lockwood, A. L.: Lung Abscess, Arch. Surg. **6**: 314, 1923. (148) Lockwood, A. L.: The Empyema Problem, Arch. Surg. **16**: 296,

1928. (149) Lord, F. T.: Diagnosis and Treatment of Abscess and Gangrene of the Lungs, With Special Reference to Operation, Internat. Clin. 2: 57, 1906. (150) Lord, F. T.: Lobar Pneumonia, Boston M. & S. J. 173: 153, 1915. (151) Lord, F. T.: Pulmonary Abscess, Boston M. & S. J. 192: 785, 1925. (152) Lord, F. T.: Diagnosis and Treatment of Acute and Chronic Non-Tuberculous Pulmonary Suppurative Lesions, Boston M. & S. J. 197: 333, 1927. (153) Lyman, H. W.: Relation of Nose and Throat Operations to Lung Abscess, J. Missouri M. A. 20: 418, 1923. (154) Lynah, H. L.: Bronchoscopic Treatment of Bronchiectasis and Pulmonary Abscess, M. Rec. 97: 215, 1920. (155) Lynah, H. L.: Series of Foreign Bodies in Bronchi and Esophagus, New York M. J. 112: 653, 1920. (156) Lynah, H. L.: Bronchoscopic Studies of Pulmonary Abscess, J. A. M. A. 77: 1548, 1921. (157) MacKenzie, L. B.: Lung Abscess, M. J. & Rec. 119: 191, 1924. (158) McKechnie, W. E.: Abscess of Lung and Liver; Simple Cure of a Chronic Case by Upside-Down Position, Lancet 1: 865, 1912. (159) McRae, F. W.: Abscess of the Lung Following Acute Lobar Pneumonia, J. A. M. A. 39: 739, 1902. (160) Maes, U.: Spirochaetal Pulmonary Gangrene, Internat. Clin. 2: 197, 1926. (161) Mandelbaum, M. J.: Postural Drainage of Lung Suppuration, New Postural Frame, the Jack-knife Position and Abdomino-Diaphragmatic Pulmonary Compression With a Review of Literature, Arch. Int. Med. 40: 840, 1927. (162) Manges, M.: Abscess of Lung, Am. J. Surg. 30: 78, 1916. (163) Marietta, S. U.: Treatment of Acute Pulmonary Abscess, J. A. M. A. 102: 1363, 1934. (164) Marshall, M. S., and Brunn, H.: Bacteriology of Pulmonary Abscess, Arch. Surg. 26: 225, 1933. (165) Maxwell, J.: Lung Abscess With Special Reference to Causation and Treatment, Quart. J. Med. 3: 467, 1934. (166) Mayer, E.: Endobronchial Treatment of Bronchiectasis, New York M. J. 108: 666, 1918. (167) Mayo, W. J.: Mortality and End Results in Surgery, Surg. Gynec. Obst. 32: 7, 1921. (168) Miller, J. A., and Lambert, A. V. S.: Treatment of Abscess of the Lung, Am. J. M. Sc. 171: 81, 1926. (169) Miller, J. A.: Lung Abscess: Some Aspects of Etiology and Medical Treatment, Laryngoscope 39: 211, 1929. (170) Miller, J.: Review of 100 Cases of Abscess of Lung, Ann. Otol. Rhin. & Laryng. 40: 1198, 1931. (171) Mix, C. L.: Lung Abscess With the Picture of Tuberculosis, M. Clin. Chicago 1: 1, 1915, 1916. (172) Moersch, H. J.: Bronchoscopic Treatment of Pulmonary Abscess, Surg. Gynec. Obst. 46: 704, 1928. (173) Moersch, H. J.: Treatment of Pulmonary Abscess by Bronchoscopy, Ann. Surg. 93: 1126, 1931. (174) Moise, T. S., and Smith, A. H.: Atelectasis in the Pathogenesis of Abscess of the Lung—Study of Spontaneous Pulmonary Suppuration in Albino Rat, Arch. Int. Med. 45: 92, 1930. (75) Moore, W. F.: Pulmonary Abscess, J. A. M. A. 78: 1279, 1922. (176) Moore, W. F.: Bronchoscopic Treatment of Suppurative Diseases of the Lungs, J. A. M. A. 82: 1036, 1924. (177) Moore, J. A.: Treatment of Pulmonary Suppuration, South. Med. & Surg. 93: 81, 1931. (178) Morlock, H. V., and Pinchin, A. J. S.: Bronchoscope in Treatment of Pulmonary Suppuration, Lancet 2: 605, 1932. (179) Morse, J. L.: Chronic, Non-Specific Infections of Lungs and Bronchi—General Introduction and Diagnoses, J. A. M. A. 87: 736, 1926. (180) Muller, G. P.: Surgical Aspects of Lung Abscess, Am. J. Roentgenol. 15: 421, 1926. (181) Muller, G. P.: Suppurative Diseases of the Chest, Surg. Gynec. Obst. 46: 193, 1928. (182) Muller, G. P.: Mortality and End Results of Operation for Abscess of Lung, Ann. Surg. 91: 361, 1930. (183) Mullin, W. V., and Ryder, C. T.: Experimental Lesions of Lungs Produced by Inhalation of Fluids From Nose and Throat, Am. Rev. Tuberc. 4: 683, 1920. (184) Myerson, M. C.: Pulmonary Aspects of Tonsillectomy Under General Anesthesia, Laryngoscope 32: 929, 1922. (185) Myerson, M. C.: Bronchoscopic Observations on Cough Reflex in Tonsillectomy Under General Anesthesia, Laryngoscope 34: 63, 1924. (186) Neuhof, H., and Hirschfeld, S.: Putrid Empyema: Ruptured Putrid Abscess of Lung, Ann. Surg. 100: 1105, 1934. (187) Nolf, P.: Fetid Spirillar Bronchitis and Pulmonary Gangrene, Arch. Int. Med. 25: 429, 1920. (188) Ochsner, A., and Nesbit, W.: A Pulmonary Abscess Following Tonsillectomy, Preliminary Report, Arch. Otolaryng. 6: 330, 1927. (189) Ophuls, W.: Acid-Proof Bacilli in Five Cases of Pulmonary Gangrene, J. Med. Res. 8: 242, 1902. (190) Overholt, R. H.: Treatment of Pulmonary Abscess by Peripheral Lung Fixation and Regional Thoracoplasty, J. THORACIC SURG. 3: 134, 1933. (191) Overholt, R. H.: Drainage and Closure of Pulmonary Abscess, S. Clin. North America 14: 1169, 1934. (192) Pancoast, H. K.: Roentgen-Ray Diagnosis of Lung Abscess, Am. J. Roentgenol. 15: 410, 1926. (193) Peters, W. H.: Hand Infection Apparently Due to Bacillus Fusiformis, J. Infect. Dis. 8: 455, 1911. (194) Pierson, P. H.: Post-Tonsillectomic Pulmonary Abscess: Medical Aspects, Arch. Otolaryng. 11: 279, 1930. (195) Pilot, I., and Davis, D. J.: Studies in Fusiform Bacilli and Spirochetes, Their Rôle in Pulmonary Abscess, Gangrene and Bronchiectasis, Arch. Int. Med. 34: 313, 1924. (196) Pinchin, A. J. S., and Morlock, H. V.: Abscesses of Lung: Their Treatment and Diagnosis, Lancet 2: 842, 1930. (197) Pinchin, A. J. S., and Morlock, H. V.: Treatment of Pulmonary Suppuration, Practitioner 127: 335, 1931. (198) Pinchin, A. J. S., and Morlock, H. V.: Further Observations on Lung Abscesses and Their Treatment, Lancet 1: 1369, 1935. (199) Portis, B.: Three Cases of Lung Abscess, S. Clin. North America 13: 1241, 1933. (200) Pottenger, F. M.: Points of Differential Diagnostic Value in Pulmonary Abscess, Bronchiectasis, Am. J. M. Sc. 158: 502, 1919. (201) Pritchard, S.: Medical Treatment of Lung Abscess, Nat. Tuberc. A. Tr. 27: 84, 1931. (202) Ransom, F. T., and McGolrick, L.: Lung Abscess Following Tonsillectomy, Brit. M. J. 2: 1021, 1933. (203) Rawson, V.: Spirochetal Abscesses of the Lung, J. A. M. A. 82: 545, 1924. (204) Rich, H. M.: Acute Lung Abscess, Am. J. M. Sc. 164: 428, 1922. (205) Richardson, C. W.: Complications of the Operation for Removal of Tonsils, Laryngoscope 23: 667, 1913. (206) Richardson, C. W.: Abscess of Lung, Laryngoscope 26: 1001, 1916. (207) Richardson, C. W.: Abscess of the Lung and the Method of Prevention in Nasopharyngeal Surgery. Ann. Otol. Rhin. & Laryng. 31: 960, 1922. (208) Robinson, S.: Surgery of Lung Disease, J. A. M. A. 59: 269, 1912. (209) Rothwell, J. H.: Bronchial Vincent's Angina, J. A. M. A. 54: 1867, 1910. (210) Sabin, F. R.: Method of Growth of the Lymphatic System, Science 44: 145, 1916. (211) Scarff, J. E.: Experimental Production of Pulmonary Abscess, Etiologic Factors, Arch. Surg. 18: 1960, 1929. (212) Schaefer, H.: Postural Treatment of Bronchial Disease, J. A. M. A. 80: 1493, 1923. (213) Schall, L. A.: Pulmonary Abscess Following Tonsillectomy, Bronchoscopic Considerations as Aid to Surgeon, Arch. Otolaryng. 11: 300, 1930. (214) Schlueter, S. A., and Weidlein, I. F.: Post-Operative Lung Abscess, Experimental Study, Arch. Surg. 14: 457, 1927. (215) Schlueter, S. A., and Weidlein, I. F.:

Surgery of Lung, Experimental Lobectomy and Pneumonectomy, Arch. Surg. **13**: 459, 1926. (216) Scudder, C. L.: Report of the Cases of Lung Abscess at the Massachusetts General Hospital Clinic, Boston M. & S. J. **171**: 523, 1914. (217) Segard, E. S.: Pulmonary Embolism and Abscess, Case Report, Mil. Surgeon **70**: 354, 1932. (218) Singer, J. J., and Graham, E. A.: Roentgen-Ray Study of Bronchiectasis, Am. J. Roentgenol. **15**: 54, 1926. (219) Singer, J. J.: Bronchography Injection of Iodized Oil, 40%, Arch. Surg. **14**: 167, 1927. (220) Smith, D. T.: Fuso-Spirochaetal Disease of Lungs, Its Bacteriology, Pathology and Experimental Reproduction, Am. Rev. Tuberc. **15**: 352, 1927. (221) Smith, D. T.: Fuso-Spirochaetal Disease of the Lungs; Its Bacteriology, Pathology and Experimental Reproduction, Am. Rev. Tuberc. **16**: 584, 1928. (222) Smith, D. T.: Fuso-Spirochaetal Disease of Lungs; Produced with Cultures of Vincent's Angina, J. Infect. Dis. **46**: 303, 1930. (223) Smith, D. T.: Diagnosis and Treatment of Pulmonary Abscess in Children, J. A. M. A. **103**: 971, 1934. (224) Solomon, N. J.: Pulmonary Abscess Treated by Artificial Pneumothorax, M. J. Australia **2**: 51, 1930. (225) Spector, H. L.: Lung Abscesses; Clinical Study, With Especial Emphasis on Conservative Treatment, J. A. M. A. **95**: 789, 1930. (226) Stern, L.: Putrid Abscess Following Dental Operations, J. THORACIC SURG. **4**: 547, 1935. (227) Stillman, E. G.: Pneumonia, J. Exper. Med. **24**: 651, 1916. (228) Stillman, E. G.: Presence of Bacteria in Lungs of Mice Following Inhalation, J. Exper. Med. **38**: 117, 1923. (229) Tewksbury, W. D.: Pulmonary Abscess, J. A. M. A. **68**: 770, 1917. (230) Tewksbury, W. D.: Pneumothorax in Non-Tuberculous Lung Abscess, J. A. M. A. **70**: 293, 1918. (231) Tewksbury, W. D.: Cases of Acute Pulmonary Abscess Treated With Pneumothorax, New York State J. Med. **110**: 849, 1919. (232) Tewksbury, W. D.: Acute Pulmonary Abscess Following Tonsillectomy Treated With Artificial Pneumothorax, Ann. Clin. Med. **4**: 347, 1925. (233) Thomson, J. A.: Pulmonary Spirochetosis, Brit. M. J. **2**: 709, 1918. (234) Tilton, B. T.: Operative Treatment of Acute Abscess of the Lung, Ann. Surg. **46**: 405, 1907. (235) Torrey, R. J.: Chronic Non-Tuberculous Pulmonary Disease, M. Clin. North America **13**: 1001, 1930. (236) Touroff, A. S. W., and Moolten, S. E.: Symptomatology of Putrid Abscess of Lung, J. THORACIC SURG. **4**: 558, 1935. (237) Tucker, Gabriel: Bronchoscopic Aspects and Treatment of Lung Abscess, Am. J. Roentgenol. **15**: 419, 1926. (238) Tucker, G.: Bronchoscopic Treatment of Lung Abscess, Am. J. Roentgenol. **15**: 419, 1926. (239) Tunnicliff, R.: Identity of Fusiform Bacilli and Spirilla, J. Infect. Dis. **3**: 148, 1906. (240) Tunnicliff, R.: Further Studies on Fusiform Bacilli and Spirilla, J. Infect. Dis. **8**: 316, 1911. (241) Tuttle, W. M.: New Method for Production of Experimental Abscesses in Dogs, Proc. Soc. Exper. Biol. & Med. **30**: 462, 1933. (242) Tuttle, W. M., and Cannon, P. R.: Effects of Local Immunization on Development of Experimental Abscesses of Lung, Arch. Surg. 30: 243, 1935. (243) Van Allen, C. M., Adams, W. E., and Hrdina, L. S.: Bronchogenic Contamination in Embolic Abscess of Lungs, Arch. Surg. **19**: 1262, 1929. (244) Varney, P. L.: Bacterial Flora in Treated and Untreated Abscess of Lung, Arch. Surg. **19**: 1602, 1929. (245) Vinson, P. P.: Foreign Bodies in Bronchi Overlooked at Bronchoscopic Examination, Report of 3 Cases, M. Clin. North America **13**: 1379, 1930. (246) Vinson, P. P.: Necessity for Bronchoscopic Examination in Distinguishing Carcinoma of Bronchus from Suppurative Disease of the Lung. Surg. Gynec. Obst. **54**: 105, 1932. (247) von Eberts, E. M.: Lung Abscess, Canada M. A. J. **1**: 1165, 1911. (248) Walker, I. J.: Acute Abscess and Gangrene of the Lung, Boston M. & S. J. **171**: 49, 1914. (249) Wall, J. S.: Lung Abscesses Following Tonsillectomy, Arch. Pediat. **48**: 687, 1931. (250) Weidlein, I. F., and Herrmann, L. G.: Abscess of Lung: Experimental Studies in Chronicity, J. A. M. A. **91**: 850, 1928. (251) Weiss, L.: Tonsillectomy in Etiology and Prevention of Lung Abscess, Eye, Ear, Nose & Throat Monthly **11**: 408, 1932. (252) Wessler, H.: Suppuration and Gangrene of Lungs, J. A. M. A. **73**: 1918, 1919. (253) Wessler, H., and Schwarz, H.: Abscess of Lung in Infants and Children, Am. J. Dis. Child. **19**: 137, 1920. (254) Wharton, L. R., and Pierson, J. W.: Minor Forms of Pulmonary Embolism After Abdominal Operations, J. A. M. A. **79**: 1904, 1922. (255) Whipple, A. O.: Etiology of Post-Operative Pneumonitis, New York State J. Med. **26**: 967, 1926. (256) Whipple, A. O.: Post-Operative Pneumonitis, Surg. Gynec. Obst. **26**: 29, 1918. (257) Whittemore, W.: Lung Abscess, S. Clin. North America **1**: 765, 1921. (258) Whittemore, W.: Non-Tuberculous Pulmonary Abscess, Boston M. & S. J. **188**: 497, 1923. (259) Whittemore, W.: Etiology and Treatment of Non-Tuberculous Pulmonary Abscess, Surg. Gynec. Obst. **38**: 461, 1924. (260) Whittemore, W., and Balboni, G. M.: Non-Tuberculous Broncho Pulmonary Suppurations; Results of Treatment by Artificial Pneumo-Thorax, Arch. Surg. **16**: 228, 1928. (261) Wilenski, A. O.: Relation of Bronchopulmonary Suppuration to Empyema, Arch. Surg. **16**: 288, 1928. (262) Williams, T. P.: Abscess and Gangrene of Lung, Lancet **1**: 336, 1926. (263) Winternitz, M. C., and Hirschfelder, A. D.: Studies Upon Experimental Pneumonia in Rabbits, J. Exper. Med. **17**: 657, 1913. (264) Withington, C. F.: Pulmonary Abscess and Gangrene, Boston M. & S. J. **138**: 220, 1898. (265) Yankauer, S.: Foreign Body in the Bronchus, Laryngoscope **28**: 433, 1918. (266) Yankauer, S.: Operative Treatment of Advanced Pulmonary Tuberculosis, Med. Rec. **97**: 1097, 1920. (267) Yankauer, S.: Bronchoscopic Diagnosis and Treatment of Lung Abscess, Laryngoscope **39**: 215, 1929. (268) Yates, J. L.: Pulmonary Abscess, Arch. Surg. **25**: 257, 1932.

THREE CASES OF RESECTION OF CALCIFIED PULMONARY ABSCESS (OR TUBERCULOSIS) SIMULATING TUMOR*

EVARTS A. GRAHAM, M.D., AND J. J. SINGER, M.D.
ST. LOUIS, MO.

THE three cases to be presented have given such diagnostic and therapeutic problems that it seemed to us that they should be recorded for the benefit of others who might be confronted with the same sort of case. This idea was strengthened by the fact that we have been unable to find in the literature any cases identical with these. From a diagnostic standpoint they all had features which strongly suggested tumors of the lung, and yet, after the removal of the lesion, in not a single case was there any evidence of a neoplasm found upon the microscopic examination of the tissue.

It is, of course, well known that calcification and even bone formation occur rather frequently in the lung, as a result of certain chronic infections, notably tuberculosis. In none of these cases, however, was there any positive evidence of tuberculosis; and, in fact, aside from the lesion itself, there was not even any presumptive evidence of that disease. In two there was marked calcification, and in one other there was a definite formation of bone. All the patients had symptoms of a chronic pulmonary inflammation which were completely relieved by the surgical removal of the lesion. We are still unable to make a positive pathologic diagnosis of the nature of the original disease in any of the cases. After their removal the lesions have shown as their principal characteristic concentric rings of fibrous tissue impregnated with calcium. Although in each case the mass itself was hard, there was found in the center evidence of liquefaction necrosis which in one case seemed to the naked eye to be definite pus. This suspicion, however, was not substantiated by microscopic examination or by culture, except in one case in which a communication with the bronchial tree had resulted in a definite infection of the mass with resulting expectoration of pus. In each case the mass had been intimately connected with the pulmonary tissue by means of bands of fibrous tissue so that it was necessary to remove part of the pulmonary tissue with the mass. The surrounding areas of lung were free from inflammation, but small regions of atelectasis were found which were thought to be due to pressure.

*From the Medical and Surgical Chest Service of Barnes Hospital and Washington University School of Medicine.

Read before the Nineteenth Annual Meeting of the American Association for Thoracic Surgery at Rochester, Minn., May 4, 5, 6, 1936.

We are emphasizing the fact that these lesions simulate tumors because we feel that the possibility of such a condition as has been encountered in our cases must always be considered when one is confronted with what seems to be a small pulmonary tumor.

CASE 1.—White, male, thirty-nine years old, was admitted to Barnes Hospital September 29, 1925, because of the shadow in his lung which had been considered by his physician to be a tumor. The patient stated that the present illness began at least as far back as 1909 and probably before that time. In 1909 he was treated

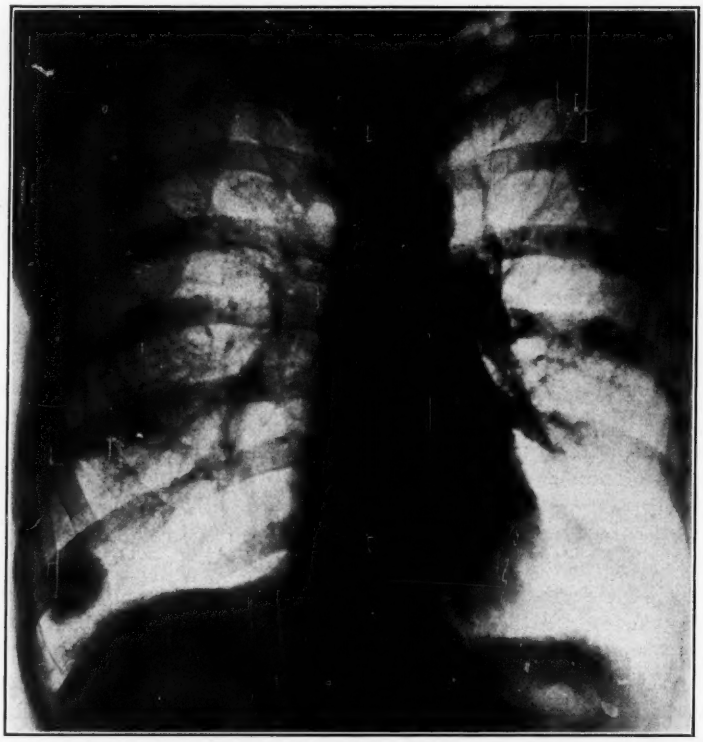

Fig. 1.—Case 1. The dense calcified mass is seen in the right lower lung field. Some irregularity of the right diaphragm.

as a tuberculous patient because of cough with sputum, night sweats, loss of weight and loss of appetite. A positive diagnosis was not made. His mother, however, had died of tuberculosis. He improved on bed rest and was well after five months. He then remained free from symptoms for about three years, at which time there was a recurrence of his symptoms and again he had bed rest for a year. Recurrences of symptoms occurred at intervals of three or four years from then on. At one time he spent five months in a tuberculosis sanatorium and at another time three months. At no time, however, was a positive diagnosis of tuberculosis made, and tuberculin tests performed in 1910 and 1921 were negative. On

admission to the Barnes Hospital his symptoms had been present continuously for about two years from the date of the last intermission, and he was daily bringing up about 500 c.c. of sputum which was frequently streaked with blood and at intervals had a bad odor.

On x-ray examination a mass was found about 3 cm. in diameter, which was roughly spherical, sharply defined and apparently partly calcified. (Fig. 1.) This was situated above the outer third of the right leaf of the diaphragm and apparently coextensive with its shadow. There was also a considerable increase of the hilus shadows on both sides. This examination was made before the use of lipiodol, and,

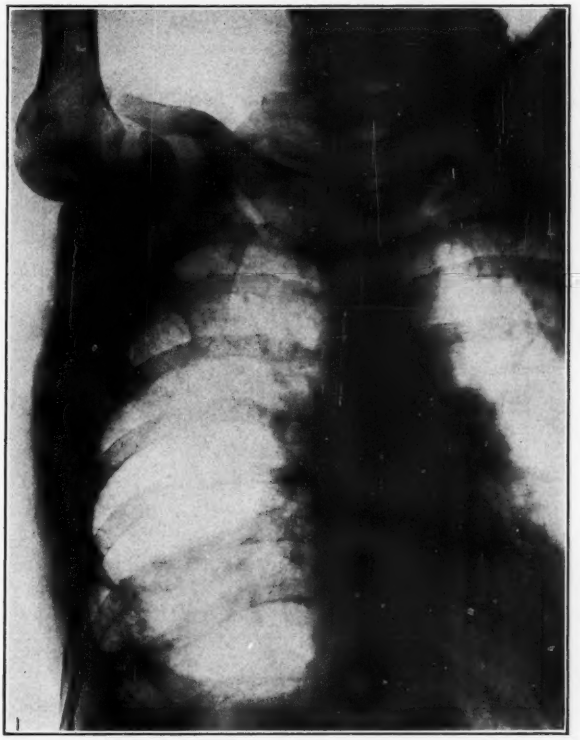

Fig. 2.—Case 1. The condition of the chest after the surgical removal of the mass.

therefore, we did not have the benefit of examination by that means although we strongly suspected the presence of bronchiectasis of the right lower lobe. On October 5, 1925, under nitrous oxide anesthesia a thoracotomy was performed after the subperiosteal resection of the eighth rib. (Fig. 2.) The mass was found to occupy the lower portion of the middle lobe of the lung. The mass, including a portion of uninvolved lung, was delivered out of the incision while a pedicle of normal lung tissue was carefully sutured with catgut to the edges of the wound. The pleura and the intercostal muscles were then sutured around the pedicle tightly in order to shut off the pleural cavity completely. Three days later the tumor was

excised with an electric cautery knife. Only one small artery in the pedicle required ligation. The patient was discharged from the hospital eighteen days after the first operation with the wound healed and entirely relieved of his symptoms.

On examination after removal the lesion was found to be a sharply limited spherical mass of chalk-white material. (Figs. 3 and 4.) Microscopic examination showed a rim of compressed lung tissue surrounding a mass of old scar tissue. (Fig. 5.) In the latter there was scattered necrotic material and spaces left by the solution

Fig. 3.

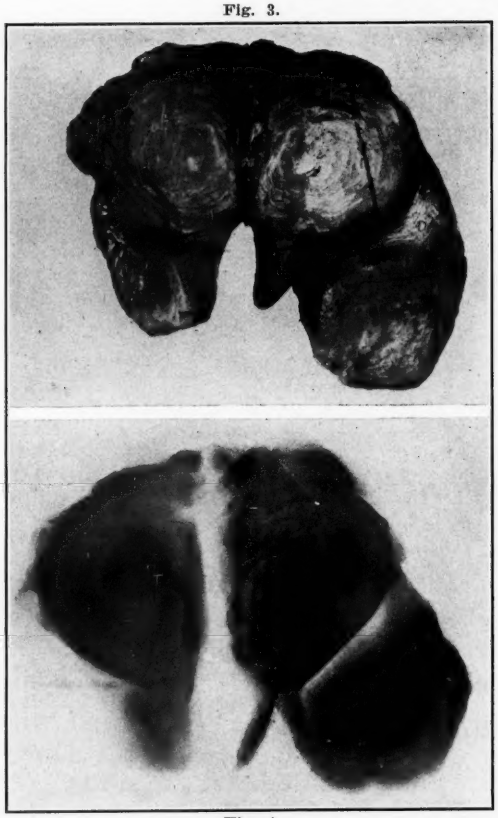

Fig. 4.

Fig. 3.—Case 1. The mass and part of the lung removed by partial lobectomy. Note the concentric rings of fibrous tissue and calcium in the cut section.

Fig. 4.—Case 1. X-ray film of the mass after its removal shows presumably the concentric rings of calcified material.

of cholesterol crystals. Around the edge of this in the old scar tissue there was a moderate infiltration of round cells. A chemical examination for calcium and phosphorus showed the following:

	CALCIUM PER CENT	PHOSPHORUS PER CENT
Ash	30.9	15.1
Dry material	8.87	4.4
Wet material	4.86	2.4

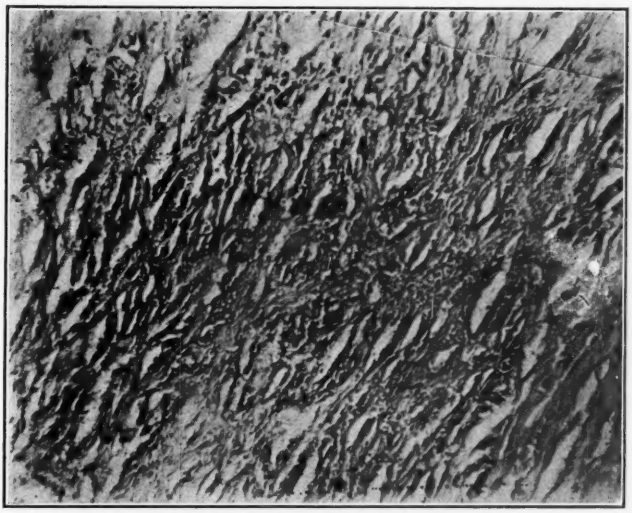

Fig. 5.—Case 1. Photomicrograph of the specimen. Fibrous tissue and calcium deposits are seen.

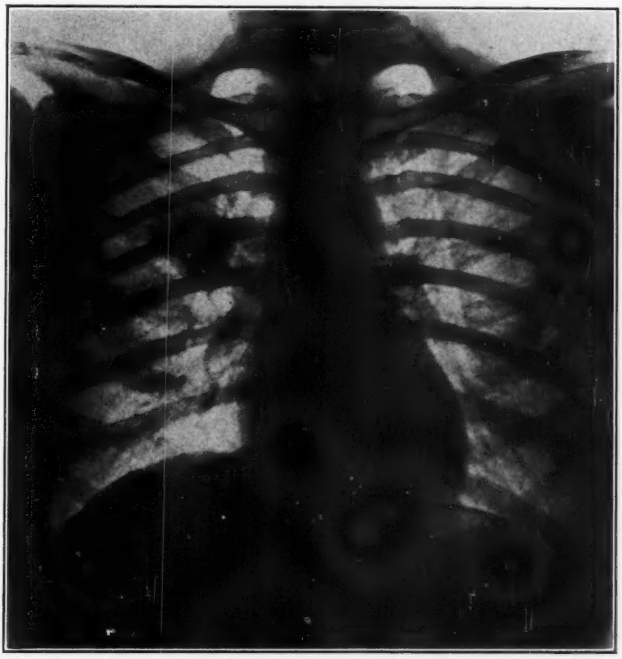

Fig. 6.—Case 2. A dense mass is seen in the right lung field over the third anterior rib. Note the dense calcification within a smaller dense area.

The conclusion was that the lesion in this case was probably the result of an old pulmonary suppuration which had healed with the formation of a considerable amount of fibrous tissue and calcification. The complete relief of the symptoms after the removal of the mass suggested that there must have been some communication with the bronchial tree. The patient has been heard from on several occasions during the eleven years which have elapsed since his operation and he reports that he is perfectly well.

CASE 2.—White, female, thirty-three years old, was admitted to Barnes Hospital on December 20, 1934. During the course of a gastrointestinal examination because

Fig. 7.

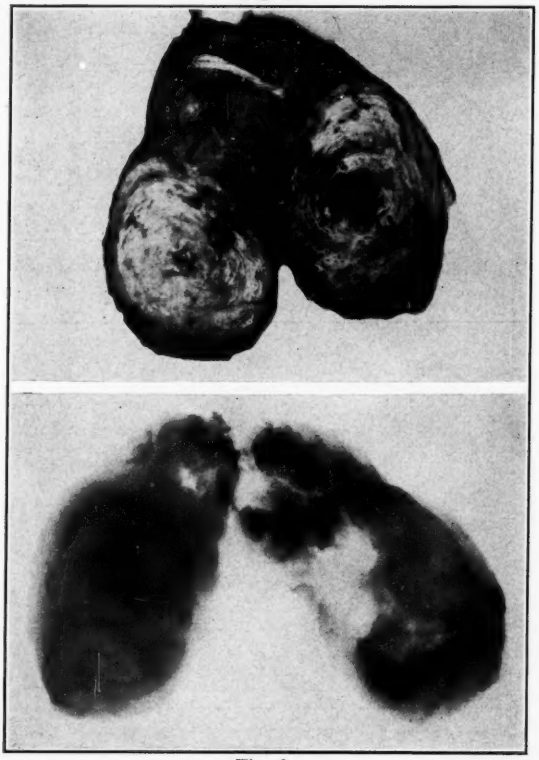

Fig. 8.

Fig. 7.—Case 2. Photograph of mass after its removal by partial lobectomy.
Fig. 8.—Case 2. X-ray film of the removed mass reveals strikingly the concentric rings of calcification.

of pain in the epigastrium, a shadow in the right lung was discovered which was thought to be a tumor. (Fig. 6.) The patient had no marked pulmonary symptoms but had complained of high backache for several months. A bronchoscopic examination by Dr. Arbuckle showed nothing abnormal. A lipiodol examination revealed some distortion of the bronchi but no bronchial obstruction. The mass appeared as a well-defined oval mass measuring about 4 × 3 cm. overlying the anterior end of the right second rib and extending into the adjacent interspaces. The lipiodol exam-

inations showed nothing of importance. The patient left the hospital for the holidays, and after her return a pneumothorax was performed in preparation for an operative removal of the mass.

On January 21, 1935, an incision was made on the right side parallel to the eighth rib with the upper angle carried upward to about the fifth rib close to the spine. After dividing the underlying muscles the sixth, seventh and eighth ribs were divided about one inch from the corresponding transverse processes. The lesion, which had been seen in the x-ray film, presented itself immediately after opening the pleura. It was on the posterior surface of the lung and formed a bridge across the interlobar fissure between the upper and the middle lobes. It was about the size of a golf ball. The posterior surface presented a peculiar appearance, in that a milk-white rectangle appeared which looked as if a piece of costal cartilage about 2 cm. long had been implanted in the lung. The regularity of the edges was very striking. Ordinary stomach clamps were placed around the mass including some

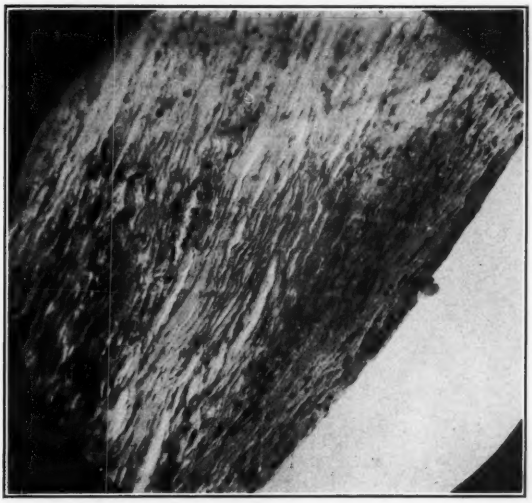

Fig. 9.—Case 2. Photomicrograph showing fibrous tissue with the laminated areas of calcium.

normal lung tissue, and it was excised with an actual cautery. The lung was then sutured with No. 1 plain catgut. No bleeding of any consequence occurred. The cut ends of the ribs were brought together by tying ligatures of No. 2 chromic catgut which had been passed through drill holes in the ribs. The incision was closed tightly in layers. The anesthesia used was nitrous oxide and oxygen administered through an intratracheal catheter.

The tumor after removal was found to measure 3.5 × 2.5 × 2 cm. (Figs. 7 and 8.) It was spherical, and on section it was found to be composed of firm elastic material which was cystic in three or four places. Some of these necrotic areas had a diameter of 3 cm. In places calcification was evident. In the center of the mass was white material which, together with the other aspects, suggested a partially calcified lymph gland. Microscopic examination showed no evidence of tuberculosis, but around the periphery of the mass was a zone of round cell infiltration, granulation tissue and atelectatic lung. (Fig. 9.) The question in this case was whether

this was an old partly healed interlobar empyema or a healed inspissated abscess of the lung. In either case it is curious that there was no previous history suggestive of pulmonary suppuration. A chemical analysis for calcium and phosphorus revealed the following:

	CALCIUM PER CENT	PHOSPHORUS PER CENT
Ash	37.3	19.2
Dry material	9.4	4.9
Wet material	4.86	2.6

The postoperative course was uneventful. The patient has been seen on several occasions since the operation, and she is now perfectly well.

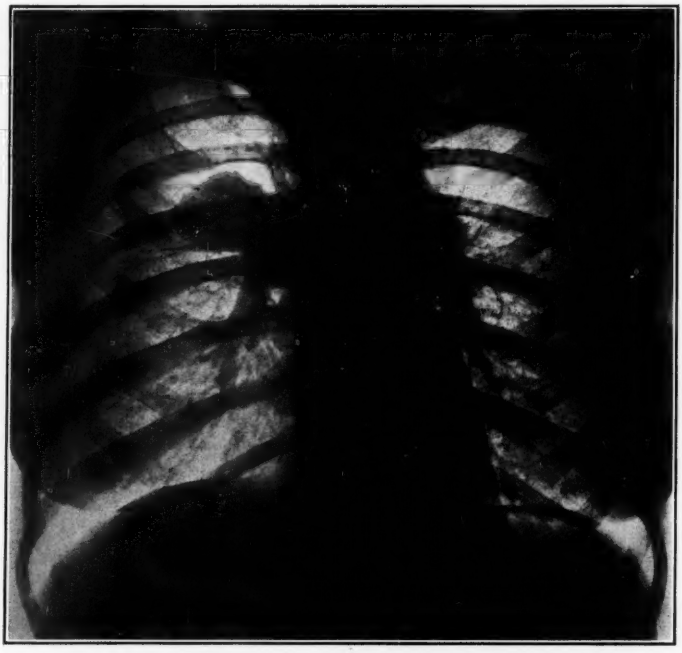

Fig. 10.—Case 3. The sharply circumscribed and dense mass is readily seen in the right lung.

CASE 3.—This case is one of the most interesting of the group. It concerns a white woman, thirty years of age. Her chief complaint was that she had had a cough with expectoration of purulent sputum for a period of one and one-half years. For about two months the sputum had been frequently streaked with blood. A diagnosis elsewhere of a mediastinal tumor was made, for which she had received three deep x-ray treatments. She was admitted to the Barnes Hospital on February 5, 1936. The onset of the present illness apparently was sudden, and at the beginning a diagnosis of flu was made. For a week she was confined to bed with fever and cough. The fever disappeared but the cough remained. The patient also was sure that during the past few months she had been aware of a distressing amount of dyspnea. The sputum which she raised was yellowish but did not have a foul odor; repeated examinations failed to show any tubercle bacilli.

The most interesting feature of the examination was a dense shadow in the region of the right upper or middle lobe. (Fig. 10.) This was considered to be probably a tumor, and an operation was advised. A bronchoscopic examination was purposely omitted because it seemed most unlikely that the mass could be reached with the bronchoscope.

On February 19 an anterior incision in the fold under the right breast was made. A mass was felt in the substance of the right middle lobe. It was firm but it seemed to be somewhat resilient as if it might contain fluid. The safest procedure seemed to be a removal of both upper and middle lobes. For purposes of ex-

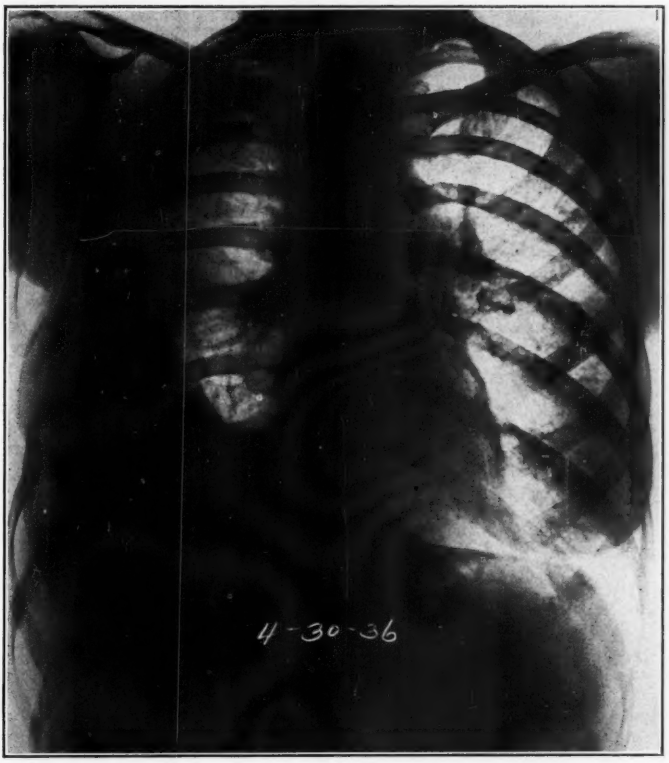

Fig. 11.—Case 3. The condition of the chest after the removal of the mass. There is a small amount of fluid in the pleural cavity which was later absorbed. Note absence of any visible deformity.

ploration the incision into the pleural cavity had been made through the third intercostal spaces after dissecting the breast upward from the chest wall. The second, third and fourth intercostal cartilages were then divided, and a rib spreader was inserted. The bronchus and vessels passing to the upper lobe were tied and divided separately as were also those of the middle lobe. The upper and middle lobes were then removed together. During the process of removing the lobes the mass accidentally burst open and about 10 c.c. of material which in appearance resembled pus escaped, although it was considered possible that the material might be mucus from a cyst or similar structure. (Fig. 11.) The thoracic cage was

closed tightly by suturing the divided intercostal cartilages together with No. 2 chromic catgut which was passed through the cartilages with a cutting needle. No drainage was used. For ligation of the pulmonary vessels and the bronchi No. 2 chromic catgut was used.

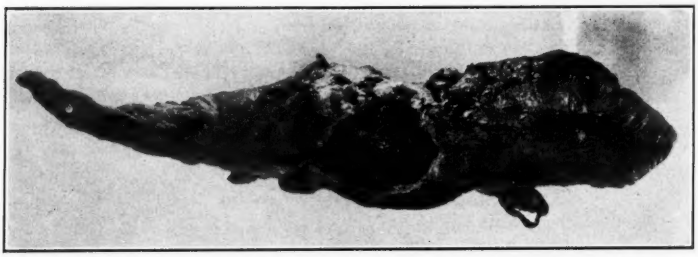

Fig. 12.—Case 3. Photograph of bisected upper and middle lobes to show the contained cavity surrounded by a dense fibrous ring. Microscopic examination revealed true bone formation in this ring, and the cavity was found to be lined with ciliated epithelium.

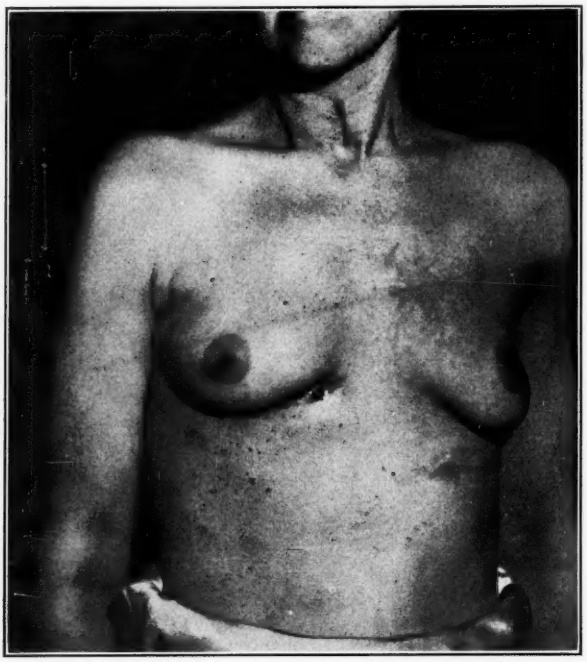

Fig. 13.—Case 3. Photograph of patient five weeks after operation. The scar is almost entirely hidden by the right breast, and there is no detectable deformity of the thoracic cage because no ribs were removed.

The patient's immediate postoperative course was uneventful. On the third postoperative day, however, she developed an alarming degree of dyspnea. It was believed that there must be some tracheal obstruction, and accordingly a tracheotomy was performed by Dr. Arbuckle. This procedure failed to relieve the dyspnea. Consequently Dr. Arbuckle passed a bronchoscope hastily through the tracheotomy

opening and was able to see clearly that the main bronchus on the unoperated side was almost completely collapsed as if from external pressure. Previous aspiration of the right pleural cavity had failed to relieve the patient's dyspnea. Accordingly, it was decided that the wound must be reopened. A most unusual complication was then discovered. The right lower lobe was swung over to the midline and was apparently attached to the bifurcation of the bronchus by recent adhesions. As soon as these were broken up by passing a finger around the right lower lobe, the patient's dyspnea completely disappeared. The wound was then closed again, but a small, tightly fitting catheter was inserted to drain the fluid which might accumulate subsequently. From that time on, recovery was uneventful, and the patient was discharged from the hospital on March 29, relieved of all of her symptoms and with the wound solidly healed.

The examination of the specimen after removal showed a cystic mass occupying what apparently had been the interlobar fissure between the upper and the middle lobes. This mass was approximately spherical and measured about 2.5 cm. in diameter. The central portion was occupied by material which seemed to be thick creamy pus. The microscopic examination of the material, however, showed no evidence of pus. Likewise no tubercle bacilli could be found within it after making numerous smears. In cutting through the mass there was resistance to the passage of the knife as if caused by a region of calcification. The most notable features of the microscopic examination were that surrounding the cavity in the center of the mass there were thin plates of true bone formation; also the cavity itself was lined with ciliated epithelium. (Fig. 12.) There was no microscopic evidence of tuberculosis and numerous sections stained especially for tubercle bacilli failed to reveal any. The rest of the lung tissue which had been removed with the mass seemed to be entirely normal on both gross and microscopic examination.

A satisfactory explanation of the origin of this mass is difficult to make. One has to consider the possibility of a congenital malformation such as a cyst or one can think of the possibility of a healed small interlobar empyema with a bronchial communication. It seems most unlikely that the condition was tuberculous, and because of its location it would seem unlikely that it was originally a pulmonary abscess.

SUMMARY

Three cases are presented in all of which there was a lesion diagnosed as a tumor associated with cough and expectoration. In all the cases the lesion in question was not neoplastic but consisted of a central portion of necrotic tissue surrounded by either calcium or bone. In all three of the patients the symptoms were completely relieved by the removal of the pseudotumor. There was no positive evidence of tuberculosis in any of the cases, and no bacteria of any kind could be found in the central necrotic portions, either on smear or by cultural methods. In Case 3 the central cavity was lined with ciliated epithelium as if the lining were derived from bronchial epithelium. In Cases 2 and 3 there was a suggestion that the original condition might have been an interlobar empyema. The final question as to whether any or all of the lesions were tuberculous or pyogenic in origin must remain unanswered. From a clinical standpoint the most important feature of all these cases was that they were all erroneously considered to be true tumors before an actual inspection and examination of them was made.

ACUTE INFECTIONS OF THE MEDIASTINUM WITH SPECIAL REFERENCE TO MEDIASTINAL SUPPURATION

HAROLD NEUHOF,* M.D.
NEW YORK, N. Y.

THE number of cases of acute infection of the mediastinum which have come under my observation during the past fifteen years indicate that such lesions are not so rare as is commonly believed. The impression became a conviction after a survey of the records of the Mount Sinai Hospital for that period. The remarks to be made are based upon a series of sixty-six cases of various forms of acute infection, suppurative and nonsuppurative, of the mediastinum. Operative findings, definitive roentgen films, or autopsies confirmed the diagnosis in the great majority of the cases. A somewhat systematic presentation of the subject appears to be warranted because the literature consists, for the most part, only of isolated case reports illustrating bizarre features or surprising recoveries. The subject can be presented only in outline form in the limited time which is available.

Classification.—A grouping of infections of the mediastinum into suppurative and nonsuppurative forms is useful only so far as the differentiation can be made with any degree of accuracy. This is easy in some cases, difficult or impossible in others. For clinical purposes suppurative lesions can be classified into the localized form, to which the term "mediastinal abscess" is attached, and the diffuse form, termed "phlegmonous mediastinitis." Regardless of the source and symptoms, mediastinal abscess and phlegmonous invasion of the mediastinal tissues are totally different lesions. The two processes thus should be clearly differentiated even though they may coexist at times.

Pathogenesis.—In this series only rarely were mediastinal infections found to have been metastatic, or due to invasion from the lung, pleura, or retroperitoneal spaces. The commonest sources were infections of the neck and traumatic perforations of the cervical and thoracic esophagus. Concerning the cervical lesions from which the mediastinal infection was derived, there were, on the one hand, cases of retropharyngeal abscess and of suppurative cervical adenitis and, on the other hand, less obvious sources such as acute pharyngitis or laryngitis, or acute nonsuppurative cervical adenitis. When mediastinal suppuration followed suppuration in the neck of nontraumatic

*From the Mount Sinai Hospital.

Read before the Nineteenth Annual Meeting of the American Association for Thoracic Surgery, Rochester, Minn., May 4, 5, 6, 1936.

origin, the lesion in the mediastinum was almost invariably a localized abscess. By way of contrast, acute nasopharyngitis was followed in most instances either by phlegmonous mediastinitis or by a spontaneously subsiding nonsuppurative mediastinal infection assumed to be a lymphadenitis. Perforation of the esophagus by a foreign body or an instrument resulted in the development of a localized mediastinal abscess or phlegmonous mediastinitis. With very few exceptions these infections were foul smelling (putrid).

The pathogenesis of mediastinal infection was obscure only in a few instances, and in most of these the mediastinitis could be termed incidental. An examination of the records makes clear the fact that a careful history with attention directed toward pathogenesis should lead often to the realization of the existence of a mediastinal infection. In this connection it may be pointed out that the mediastinal space is not inert, but is under negative pressures. Indeed, one can picture suction from the mediastinum as a force which is applied to any infective focus which is situated at the superior aperture of the thorax.

Bacteriology.—The organism most commonly cultured from nonperforative suppurations of the mediastinum was the hemolytic streptococcus.

Pathology.—The mediastinum can be infected from the neck in three ways: by lymphangitis, by cellulitis, or by the direct downward extension of an abscess. A periesophageal abscess appears to be the first phase after perforation of the thoracic esophagus. In any event, the suppurative lesion of the mediastinum is usually fully developed when encountered at operation or at autopsy. For this reason the very early pathologic features cannot be described. In all cases of mediastinal abscess there is well-defined limitation, but usually no encapsulation by an inflammatory envelope, as in abscesses elsewhere. Indeed, there may be little or no surrounding reaction and no adjacent lymphadenitis. In most instances mediastinal abscess appears to have evolved by direct extension. When a gap exists between the source of infection and the mediastinal abscess, a suppurative mediastinal lymphadenitis is postulated. It could be recognized as such, however, in only a very few instances. The most striking type of mediastinal abscess is that which erodes into adjacent adherent lung and then perforates into a bronchus. This important variant apparently has been overlooked, yet is by no means uncommon (eight times in our series). The lesion was situated in the posterior mediastinum in all cases. A pathologic feature of almost all suppurative lesions of the posterior mediastinum is the deep situation of the focus. This is of surgical importance, since there is usually little or no contact between the abscess and the thoracic cage. The pleura adjacent to the lesion is adherent, but adhesions rarely extend superficially to a site

at which they could be traversed for the operative entry of the abscess. This held true even in unusually large abscesses. A large collection of pus originating in the posterior mediastinum need not remain confined to that region, for there are no aponeurotic barriers between the compartments. In one operative case the abscess extended from the posterior into the middle and anterior mediastinums, the anterior limit being more than 25 cm. from the posterior chest wall. The size which mediastinal abscesses can attain is illustrated by the finding, at autopsy, of 350 c.c. of pus in the case of an eighteen-month-old child.

Turning to phlegmonous mediastinitis, a significant characteristic was the coexistence of bilateral suppurative pleuritis in almost all instances in which the posterior mediastinum was involved. In contrast, bilateral suppurative pleuritis did not complicate any of the cases of mediastinal abscess in the series. Obviously, these opposed features are of prognostic importance, especially when the frequent coexistence of pericarditis with bilateral pleuritis is recalled. In several instances phlegmonous mediastinitis extended downward to involve the retroperitoneal tissues and peritoneum. It is of interest to note that the peritoneal invasion was due to direct extension in a septic patient with scattered metastatic foci.

Mediastinal abscesses may dissect their way into the pleural cavity or into extrapleural planes. Surgical drainage may have been instituted on the assumption that the lesion was an empyema. If a sinus persists, its connection with the mediastinum may be demonstrated, even after many months, by the injection of radiopaque fluids. A tendency for communications with the mediastinum to persist was found in other cases. Similarly, an esophageal communication was noted in some instances when healing of a perforation apparently had occurred. Mention should be made of the fact that direct pleural entry and infection may follow instrumental perforation of the esophagus without an intermediate invasion of the mediastinum.

Reference has not as yet been made to the pathology of acute nonsuppurative mediastinal lesions, and little will be said on this obscure subject. If the lesion subsides spontaneously, absence of suppuration can be assumed. In a few instances the clinical features and the roentgen films supported the assumption of a mediastinal lymphadenitis. In two cases the evidence pointed strongly to a suppurative mediastinal lesion but spontaneous subsidence occurred.

Finally a few comments on the type termed "incidental mediastinitis" may be made. Nonsuppurative or suppurative invasion of the mediastinum can occur in sepsis or, in transit, in descending infection from the throat or ascending infection from the retroperitoneum. In the six cases in this series, the lesion presented no clinical features and was a postmortem disclosure.

Clinical Manifestations.—The great variety of clinical manifestations which were encountered defy any very systematic classification. At the outset two statements must be made. First, the textbook picture of suppurative mediastinitis was seen only rarely. I refer to the description of recurrent chills, high fever, profound prostration, severe toxemia, typhoid state, etc. Second, in only one instance in the series, an abscess of the anterior mediastinum under great tension, was there evidence of interference with circulation. This again is in contradistinction to descriptions of compromised vascular channels and nerves as signs of mediastinitis. Because of the possibility of error in diagnosis at operation, the clinical manifestations of cases of mediastinitis which were verified by autopsy were specially analyzed, and confirmation of the two statements was obtained.

The cases of suppuration in the mediastinum in the series can be classified clinically into fulminating, moderately severe, and relatively mild types. The fulminating cases were usually fatal (within a few days), presenting either the classical picture of overwhelming sepsis which was, as stated, but rarely seen, or, more commonly, dysphagia, dyspnea, and, later, the picture of a severe pneumonia. The etiology was a throat infection, with or without a retropharyngeal abscess, or esophageal trauma. Phlegmonous mediastinitis was found at autopsy in most of the cases, but the important point is that localized abscesses were encountered in a few instances, and may therefore have been amenable to operation. Cases of moderate severity were also of nontraumatic or of traumatic origin. If an abscess did not exist, or, if present, had not been adequately evacuated, the infection was fatal in all cases. Under these circumstances the disease lasted from a week to a month or longer. Beginning with sore throat, fever, a chill in a nontraumatic case, and with pain in the neck and chest, dysphagia, and fever in a traumatic case, the course was characterized by moderate toxemia and dysphagia. Of great significance was the occurrence of a deceptive remission in several cases. In some, the period of apparent improvement was definite and clear-cut, with subsidence of fever and dysphagia, disappearance of preexisting spinal percussion tenderness, and return of a feeling of well-being. There were three cases in which operation was deferred because a remission, interpreted as spontaneous subsidence, had set in. Indeed, operation on the mediastinal abscess was performed in two of the three patients only because of x-ray evidence of an increasing lesion. The fact that postmortem examinations in these two cases disclosed additional irremediable infection does not prove that it existed from the outset. The danger in interpreting a remission as progress toward spontaneous cure is particularly stressed.

In another clinically important group of cases, already referred to, a so-called remission was synchronous with the expectoration of foul

pus. There should be no difficulty in recognizing these cases. The etiology was penetration of the esophagus in six of eight cases. The remission was followed by a mildly toxic course with cough and foul expectoration (in the case of esophageal perforation) as in pulmonary abscess, or, on the other hand, an acute fulminating picture set in after partial subsidence of symptoms. In one case recovery followed mediastinotomy and drainage performed on a patient who was septic and delirious at the end of a two weeks' illness.

In cases with relatively mild clinical course a suppurative lesion in the mediastinum was always a well-localized abscess. There was an antecedent abscess in the cervical region, and fever without discernible local cause after the abscess had been drained. Dyspnea and occasional cough called attention to the chest, and the substantial globular shadow found on the x-ray film was unexpected in all cases.

Reference should be made to some varieties of suppuration in the mediastinum in which clinical manifestations referable to that region are lacking. "Incidental mediastinitis" has already been mentioned. There were a number of instances in which involvement of the mediastinum could be recognized only at operation, classified as follows: (1) prevertebral abscess perforating into the pleura, producing an infrapulmonary empyema; (2) abscess of the lung in the paravertebral region invading the mediastinum; (3) empyema perforating into the mediastinum; (4) abscess about the cervical esophagus (following traumatic perforation) invading to a limited extent the posterior superior mediastinum. To these should be added instances in which the mediastinum was traversed but not laid open by instrumental perforations of the thoracic esophagus entering directly into the pleural cavity. Finally, mention has already been made of "incidental mediastinitis" and sinuses of the chest wall which can be proved to have been derived from a preexisting mediastinal infection only by x-ray studies.

The symptomatology outlined above refers to suppuration in the posterior mediastinum. There were only a few cases of invasion of the anterior mediastinum in our series. In these the absence of pleuritis, often seen at autopsy in posterior mediastinitis, is noteworthy. Another point of interest, already referred to, is that the only example of clinically apparent vascular blockade (inferior vena cava) was seen in a long standing tense abscess of the anterior mediastinum.

Little will be said concerning nonsuppurative acute infections of the mediastinum because there were only a few cases in which the roentgen findings were adequately confirmatory. Furthermore, the comments that can be made are not constructive. Three cases were characterized by a febrile course of several weeks' to months' duration with symptoms only vaguely referable to the chest. In two cases, on the

contrary, the clinical manifestations, physical examination, and roentgen features were identical with those noted in mediastinal abscess of moderately severe grade. Both had anterior displacement of the trachea and positive chest films. The only reason they were not operated upon was that a policy of waiting "still another day" happened to be assumed in each. Another case should be mentioned because the logic again favored operative intervention. The patient presented signs of a periesophageal invasion in the neck and some extension down into the mediastinum four days after a chicken bone perforation of the cervical esophagus. The symptoms, febrile course, and x-ray films suggested suppuration, but spontaneous subsidence (without appearance of pus) occurred.

Physical Examination.—Except for signs of pleural effusion or of pneumonic infiltration, the physical examination of the chest was essentially negative in most cases. As an exception, there can be cited a child with a large abscess in the superior mediastinum in whom there was dullness to flatness on percussion over most of the upper chest. Normal swallowing sounds were heard in the few cases in which the test was made. Furthermore, normal passage of a barium meal was noted in proved mediastinitis. Percussion tenderness over the spine, well localized in several cases, was found in posterior mediastinal infections. In two instances, however, this sign disappeared during the spread of the infection. Indeed, the waxing and waning of physical signs renders them singularly unreliable as guides to the situation and the state of the mediastinal infection. When the latter was derived from the cervical region, definitive information was usually available in an examination of the neck. In traumas to the cervical esophagus, infiltration and tenderness were usually present, and in some instances crepitation was noted. The most important evidence, however, of mediastinal extension of a deeply situated lower cervical inflammation or abscess is available from an examination of the trachea. The latter may or may not be pushed to the right or left, but is always displaced forward so that the finger cannot be dipped easily into the suprasternal hollow. In cases in which tracheal mobility was sought for, the trachea was found to be fixed more or less completely and pain was induced by the test.

Radiographic Features.—In the literature mention is made of the occasional typical film of mediastinal lymphadenitis or of mediastinal abscess, but the information which can be obtained otherwise is not brought out. The statement that the roentgenogram often is negative in acute mediastinitis is essentially correct. A more accurate statement would be that the film may not be informative in phlegmonous mediastinitis or in a mediastinal abscess situated near the diaphragm. The fact is that the x-ray film was positive in this series in almost all

the cases in which a film was taken. The abnormalities in the film were not always striking; in general, they were in keeping with the pathologic process. The more or less lobular shadows of lymphadenitis and the globular shadow of abscess have been mentioned. A localized widening of the mediastinum was seen frequently and was present in abscess as well as in phlegmonous mediastinitis. The most impressive film is probably that of a fluid level capping a shadow in the mediastinal zone. In my opinion, a fluid level across the midline is strong presumptive evidence of a mediastinal lesion. I recall only one instance in which the fluid level of a proved pyopneumothorax straddled the midline. Of course, reference is not made to various other pleural lesions (as in tuberculosis or trauma) in which the mediastinum may be herniated by a collection of air and fluid under tension. The fact that a fluid level existed was deemed indubitable proof of perforation of the mediastinal abscess into lung and bronchus, and confirmation was obtained in all cases at operation or at autopsy. The roentgen film of air in the mediastinum (or in the soft tissues) without fluid level was evidence of trauma and not of a gas-producing infection, in this series. The lateral films of the neck demonstrated a widened retrotracheal space with or without fluid level in infections descending to or into the mediastinum. Such films were of signal clinical importance and were particularly significant when successive ones showed increase or decrease in the widened retrotracheal shadow. In lateral films of the chest there was noted an anterior bowing of the intrathoracic portion of the trachea in two cases of posterior superior mediastinal infection. In one increased bowing was seen in successive films and demonstrated thereby the increase in the mediastinal lesion. If anterior displacement of the thoracic trachea as a whole can be identified in the lateral chest film, it constitutes additional evidence of a posterior mediastinal lesion.

Diagnostic Problems.—Many errors in diagnosis as well as in interpretation of the course of a mediastinal infection were made in the series. Some have been indicated. Reference has been made to the occasional difficulty in differentiation between suppurative and nonsuppurative lesions. In a few cases mediastinitis was not suspected until an x-ray film was taken or until an autopsy was performed. The diagnosis was pneumonia in three cases until the roentgen film revealed the nature of the lesion. A surprising fact was that there were three cases in children in which retropharyngeal abscess was suspected, examined for, but could not be felt through the pharynx. Again, there were instances in which the larynx or trachea instead of the esophagus was thought to be the site of foreign body lodgment or of laceration, despite persistent dysphagia. The judicious use of the bronchoscope or the esophagoscope might reduce the incidence of

erroneous diagnosis in special cases. The formidable list of errors above given indicates, however, that there is room for great improvement. The latter will be brought about when histories are carefully taken and analyzed, and due weight is given to symptoms. For example, with an abscess in the cervical region and the development of dysphagia, the first phase of a mediastinal infection should be thought of and a lateral film of the neck should be taken. If, in addition, there are symptoms and signs referable to the chest, pneumonia should not be considered in the diagnosis until a mediastinal infection has been excluded. Above all, a laceration of the esophagus, so often the precursor of mediastinitis, must be suspected whenever sharp pain follows immediately upon instrumentation of the cervical or thoracic esophagus. The situation calls for a prompt roentgenogram and an equally prompt esophagoscopy. In general it can be said that the correct diagnosis of a potential or of an existent mediastinal infection will be made more often if the erroneous impression of its rarity is dismissed and the likelihood of its development under indicated circumstances is borne in mind.

Indications for Operation.—Although this paper deals with the subject of acute infections of the mediastinum, the most important operative indication, in my opinion, is concerned with the situation which frequently precedes the mediastinal infection. Instrumental or foreign body penetration of the cervical or thoracic esophagus was the etiology in almost 50 per cent of the cases. Operation was not advised in some, but was performed in others when a mediastinal abscess was diagnosed. In no instance was operation performed at the time of perforation. Nevertheless, an analysis of the cases leads to the conclusion that an external operation, consisting in exposure of the tear in the esophagus, and drainage to that area, was indicated urgently, regardless of the site of the lesion. That spontaneous recovery or recovery after rupture of a periesophageal abscess into the lumen of the esophagus may occur is well known. A consideration of the traumatic area after the esophagus has been perforated, the inability to control the spread of infection, the insidious manner in which a serious mediastinal infection may develop, the difficulties in diagnosis, and, above all, the high mortality of mediastinitis should lead to the decision to operate in all cases. The decision should be made even though the patient is essentially symptom free at that time.

Infection in the cervical region of nontraumatic origin was the source of the mediastinal infection in about one-fourth of the cases in the series. Cervical abscesses were present in seven cases. Special reference should be made to the small but important group of cases of retropharyngeal abscess in children mentioned in the discussion on diagnosis. With suspicion aroused by the clinical course, a lateral roentgenogram of the neck will go far toward establishing the diag-

nosis. Exploration through an external incision is then warranted even though the abscess cannot be discerned by digital examination through the mouth.

Concerning the indication when a mediastinal infection exists, operation should be proceeded with, no matter how extensive the lesion may be or how septic the patient, if there is any likelihood of encountering a collection of pus. Only under unusual circumstances can one be sufficiently sure that the lesion is a phlegmonous mediastinitis and therefore surgically irremediable. Operation is indicated also when there is doubt as to the suppurative or nonsuppurative nature of the mediastinal infection. In this series there were three instances in which operation was seriously considered, not performed, and recovery ensued. They are more than counterbalanced, however, by at least six cases of apparently remediable abscesses in which operation was either withheld or performed as a last resort. Finally, operation in the nature of an exploratory mediastinotomy is indicated when a mediastinitis is suspected on etiologic and clinical grounds, but cannot be established by physical examination or roentgenogram.

Operative Procedures.—In operations for lesions about the cervical esophagus, a precise technic should be employed whether or not an obvious infiltration or mass is palpable. The following technic has been found serviceable for periesophageal lesions situated laterally or prevertebrally: A long incision is made along the anterior border of the sternomastoid. The carotid sheath is exposed and retracted laterally, care being taken to avoid opening the sheath. Ribbon muscles and anterior belly of the omohyoid are severed if necessary for exposure. The lobe of the thyroid gland is elevated and retracted mesially, vessels passing across the field to the gland being ligated. With deep retractors in place, further dissection along the lateral wall of the esophagus is carried out under good illumination. In laterally situated abscesses, an inflammatory membrane is soon encountered and is traversed to enter the abscess. The esophagus should be lifted forward for the entry of an abscess between it and the vertebral column. In either case the abscess must be laid open to its full limits. When an extension from the neck to the mediastinum is suspected, the lowest portion of the abscess cavity is visualized fully because the suppurative tract will extend from this area sharply downward and backward to enter the posterior superior mediastinum. The marked convexity of the curve (convex forward) is a striking feature. If there is extension of suppuration into the mediastinum, the tract can be laid open adequately from the neck as far as the body of the second or third or possibly the fourth dorsal vertebra. Drainage through the neck will not be adequate below this level, in my opinion (based on several experiences), and a posterior mediastinotomy should be added. With special reference to the cases of perforation of the

cervical esophagus, it can be said that operation should never be considered completed by the drainage of the infection of the neck when there are clinical signs of mediastinitis. The entry of the anterior mediastinum through the neck will probably be indicated only for infections following thyroidectomy and does not present any technical problem.

The operative approach to the posterior mediastinum should be based on painstaking efforts at localization of the lesion by physical signs, x-ray examination, and esophagoscopy in some of the perforative lesions. Patients are generally too ill to withstand the formal wide opening of the posterior mediastinum which has been advocated. Indeed, any unnecessary dissection of mediastinal spaces invites a spread of infection to that extent. The frequent absence of parietal pleural adhesions has been indicated. It is therefore questionable whether exploratory puncture should be employed when the evidence points to a deeply placed collection of pus. In one case in the series pus was not encountered until the aspirating needle penetrated to a depth of 12 to 14 cm. from an intercostal space. The incision should be placed paravertebrally and vertically so that it can be extended upward or downward. A section of the selected rib directly adjacent to the transverse process is subperiosteally excised, and then a portion of the transverse process is removed. Parietal pleura is stripped away, after which one rib and transverse process above and one below are similarly removed. Excision of three ribs sufficed in this series. The effort was made to enter the abscess without entering the pleura. This failed in most of our cases. The pleural cavity was entered six times. Since the statement has been made that death ensues from fulminating empyema under such circumstances, the results in these cases are of interest. In four, drainage was performed in one stage after the pleural cavity was packed off or the lung sutured into the pleural opening. All recovered without the development of empyema. A two-stage operation was done in two cases. Both patients died. Death was due to other causes in one of these; an empyema was a contributing cause in the other. The conclusion should, of course, not be drawn from this small series of cases that the pleura can be traversed with impunity for the drainage of a mediastinal abscess. In my limited experience, I was unable to avoid an opening in the pleura when the abscess was approached. If the abscess were situated strictly in the median line the overlying pleural reflection could be followed for an extrapleural entry. However, most mediastinal abscesses appear to project markedly to one side or the other. The technic can undoubtedly be improved. The point to be made here is that every precaution should be taken to shut off the free pleural cavity if it has been traversed for the entry of the mediastinal ab-

scess. Whether this should be done by packing, suture, or suture of lung into the opening depends on the situation which is encountered and the views of the operator.

The opening into the mediastinum should be adequate for satisfactory investigation, and exploration should not be discontinued until there is full assurance that a mediastinal abscess does not exist. In two cases the lesion was missed; one of these was a mediastinal abscess. As soon as an abscess is entered retractors should be put in place, for the walls of the abscess cavity tend to collapse. The interior of the cavity should be illuminated with a sterile light in order to see and open up all recesses and to learn the full depth of the lesion. A complete operation should be performed whenever possible, even if the patient is profoundly prostrated or septic, because residual collections of pus will lead to the continuation of the septic or toxic state. In one instance the suppurative tract was followed from the posterior into the middle and then into the anterior mediastinum. In another case a tract was followed for a considerable distance in a vertical direction. After the abscess cavity is fully visualized, its interior is snugly packed with gauze to avoid premature collapse.

There is a special problem in the mediastinal abscess which ruptures into the lung. The mediastinal may be much smaller than the pulmonary component. In one case the esophageal laceration and the mediastinal abscess had occurred long before the time of operation, and drainage via the bronchus was profuse. The mediastinal portion of the suppurative process was found to be relatively insignificant. The operative approach may be made directly over the abscess in the lung or via the abscess in the mediastinum. Proceeding from the latter, the opening into the lung was found to be large in all our cases. The overlying lid of lung was excised until the cavity in the lung was fully unroofed. The operative treatment of the pulmonary lesion was not regarded as completed until a bronchial fistula was demonstrable.

Results.—The 66 cases may be divided into those in which no operation was performed (34) and those in which some operative procedure was carried out (32). In the nonoperative group there were 9 recoveries and 25 deaths. Of the recoveries there were 5 cases of mediastinal adenitis or cellulitis, 2 of mediastinal cellulitis related to periesophageal abscesses draining into the esophagus, and 2 of incidental mediastinitis. Of the deaths, phlegmonous mediastinitis was present in 11 cases, incidental mediastinitis in 7, and mediastinal abscess in 4 cases. In two patients in whom there was no postmortem examination the lesion was either an abscess or a phlegmonous mediastinitis. There was one case of an infected hematoma of the mediastinum.

Various operative procedures were employed in the group of 32 cases. They are listed as operative cases even though a number of the operative procedures were limited to emergency tracheotomy, removal of foreign body, or drainage of empyema. Only personally operated cases will be reported upon. Twenty-two operations were performed with 15 recoveries and 7 deaths. The operations followed by recovery were for mediastinal abscess, 4 cases; mediastinal abscess complicated by lung abscess, 3 cases; lung abscess complicated by mediastinal abscess, 1 case; abscess of neck penetrating into mediastinum, 2 cases; mediastinal abscess complicated by empyema, 1 case; empyema complicated by mediastinal abscess, 2 cases; mediastinal sinus, 2 cases. The operations followed by death were for mediastinal abscess with phlegmonous mediastinitis, 1 case; mediastinal abscess complicated by lung abscess, 2 cases; abscess of neck complicated by phlegmonous mediastinitis, 1 case; laceration of esophagus penetrating into pleura, 1 case; negative exploration in perforated esophagus with mediastinal abscess, 1 case; abscess in the anterior mediastinum, 1 case. Of the patients who died, a postmortem examination seemed to prove that recovery might have ensued in three instances with earlier operative intervention or better technic.

SUMMARY

1. The impression that acute infections of the mediastinum are rare is erroneous. Unless the likelihood of their evolution under given circumstances is borne in mind, lesions amenable to surgical therapy may be overlooked. A study of sixty-six cases of suppurative and nonsuppurative mediastinal infections is presented. For clinical purposes suppurative lesions are classified as localized (mediastinal abscess) and diffuse (phlegmonous mediastinitis).

2. The commonest causes of suppurative lesions are traumatic perforations of the esophagus and infections in the cervical region.

3. Among pathologic features of posterior mediastinal abscess are limited inflammatory reaction, absence of superficial pleural adhesions, and the abscess which ruptures into the lung and bronchus. Suppurative pleuritis is characteristic of phlegmonous mediastinitis.

4. Acute mediastinal infections can be divided, clinically, into fulminating, moderately severe, and relatively mild forms. Textbook pictures are rarely seen. Physical signs are generally unreliable, being inconstant, and variable in individual cases. However, examination of the neck, in cases of mediastinal infection derived from the cervical region, yields important information. The roentgen film of the chest is usually positive, and that of the neck offers decisive information when low cervical infection is the source of the mediastinitis.

5. Immediate operation is indicated when perforations of the cervical or thoracic esophagus have occurred. When mediastinitis has developed, operation is indicated if there is any evidence of localized suppuration. Exploration is justified under certain circumstances even in the absence of positive evidence of a mediastinal abscess. Recovery may follow operation even in far advanced cases.

6. A technic is described for the approach to a cervical periesophageal abscess; it serves also for drainage of the upper posterior mediastinum. In posterior mediastinotomy the site of the incision depends upon roentgenologic localization of the level of the lesion. The free pleura was traversed in most instances. With proper management of the pleural opening, empyema need not ensue after a one-stage operation.

7. Death occurred in all cases of mediastinal abscess which were not operated upon. Recovery followed operation in four or five cases of mediastinal abscess. Of eight additional operative cases which were complicated by lung abscess, empyema, or phlegmonous mediastinitis, four patients recovered and four died. Thus mediastinal abscess is a surgically remediable lesion in which the results of operation should be good unless the abscess has become complicated as a result of delay.

DISCUSSION OF SYMPOSIUM ON SUPPURATIVE LESIONS OF THE CHEST: "NONTUBERCULOUS ABSCESS OF THE LUNG" BY CUTLER AND GROSS; "TREATMENT OF LUNG ABSCESS" BY ALLEN AND BLACKMAN; "THREE CASES OF RESECTION OF CALCIFIED PULMONARY ABSCESS SIMULATING TUMOR" BY GRAHAM AND SINGER; AND "ACUTE INFECTIONS OF THE MEDIASTINUM WITH SPECIAL REFERENCE TO MEDIASTINAL SUPPURATION" BY NEUHOF

DR. HOWARD LILIENTHAL.—I want to compliment Dr. Neuhof on his fine presentation, and I wish to comment very briefly on one or two points.

First of all, the medical men are afraid to have the mediastinum operated upon, and we should teach them they need not be afraid. Often the patient will die if he is not operated upon, and he cannot do worse if he is. But, as you have heard, they do not all die; mediastinotomy has always been a favorite operation of mine. If progressive mediastinitis is suspected, it is usually safer to operate than to wait.

In two cases of lung abscesses, I have opened deep in the lung, by the posterior mediastinal approach, and in both instances I have had recoveries.

There is one other point which Dr. Neuhof has not brought up, probably because he has not tried it, and that is something I have just begun to use now, which I consider will be of great value, and that is the employment of infra-red photography, days apart, if you can wait so long. Except in the most acute cases, a few pictures by infra-red taken a few days apart may be of diagnostic importance because the veins will show beautifully, and the increase in their number and enlargement as seen in the upper thorax will be additional evidence of mediastinal pressure. It costs little, is not so expensive as roentgenography, and is very easily done. I think all our hospitals should be equipped with an infra-red apparatus.

About lung abscess, you have seen all these beautiful statistics, and yet when you encounter a patient with a lung abscess, you must decide what to do with that patient. I have always believed that a progressive abscess, especially a rapidly progressive one, must be operated upon. If it is static or very slowly progressive, then you may think and look up statistics if you think best.

One epigram about lung abscess: Diagnosis is a science; treatment is an art.

DR. LEO ELOESSER.—It has been the fashion to belittle the medical man; but in considering these statistics as well as almost all other statistics on the surgical treatment of pulmonary abscess, I think we acknowledge that there we have something rotten in Denmark. Medical statistics are better than our own. We constantly pat ourselves on the back; we give authoritative directions what to do, but the fact remains that our statistics for pulmonary abscess are very, very bad. The time has come when we should try to do better. Dr. Allen's paper may serve to illustrate my meaning. He gave some figures, the results of which are bad; in spite of that he draws what seems to me to be an unwarranted conclusion as to what we should do.

Dr. Cutler mentioned that he excluded patients with spirochetal gangrene. I should like, not in the spirit of criticism but of inquiry, to inquire what he means by an abscess, and what is his distinction between spirochetal abscess and spirochetal gangrene.

He mentions that a number of his abscesses are spirochetal in origin.

I think that perhaps one might do a little better by abscesses if one divided the operations regularly into two stages. At the first stage a subcostal compression is done by pushing gauze between the ribs, without resecting them; at the second stage the ribs are divided, the gauze is removed, and the abscess is opened with a cautery. Aspiration of the contents of the abscess into other parts of the lung at the moment when the abscess is opened is one of the serious dangers. This may be avoided: (1) by having the patient lie or sit with the affected side *down*, over the edge of the table if need be, with the surgeon working from underneath; (2) by making a small opening at first and enlarging it only after the patient has expelled the contents of the abscess; and (3) by keeping the patient under positive pressure from the time the abscess is opened until it is sealed off by gauze packing. I should like to recall to your attention Iselin's excellent paper which we heard a few years ago. He drew the conclusion that abscess empyemas differ accordingly as the abscess ruptures into a virgin pleura, or makes adhesions for itself when it ruptures. The latter seems to be not a bad complication. I agree with Dr. Cutler that exploratory puncture is a dangerous maneuver in pulmonary abscess.

Dr. Graham may remember that in 1923, during the meeting of the American Medical Association in San Francisco, he, together with Dr. Bloodgood, was kind enough to see a patient for me who presented a picture precisely similar to the ones he showed here. That patient had an abscess, not a tumor, as we thought at the time. I have seen four cases like Dr. Graham's. One of them proved to be an encapsulated pleurisy, somewhat like his. One of them proved to be a communicating empyema which was globular and encapsulated in an intralobar fissure; in one case the diagnosis is still unknown; the patient had no symptoms and was not operated upon.

Dr. Neuhof's paper warranted more time than he was given; I shall await its publication with much interest.

DR. ETHAN FLAGG BUTLER.—Mediastinal abscesses due to esophageal perforation will show an abundance of mouth organisms. If spirochetal organisms are present, these abscesses after surgical drainage may be advantageously irrigated with a weak solution of neoarsphenamine. I have employed a 0.3 per cent aqueous solution and noted dramatic improvement.

A large proportion of nontuberculous lung abscesses will come to surgical drainage. One may then choose between simple drainage and radical drainage. By simple drainage is meant the resection of one or two ribs directly over the abscess cavity and the establishment of drainage through this small opening in the chest wall. By radical drainage is meant the wide approach by a large skin muscle flap, the resection of portions of several ribs and the removal of the soft structures of the chest wall proper so as to expose the entire abscessed area and the surrounding zone of pneumonitis. In this radical operation the skin muscle flap is left wide open until the pulmonary suppuration has been controlled. It is then closed by secondary suture with due regard to anatomical structures.

In my experience somewhat over fifty cases have come to one or the other types of surgical drainage. Of those subjected to simple drainage, 62.3 per cent eventually recovered and 37.7 per cent ultimately died. Of those subjected to radical drainage, 65 per cent eventually recovered and 35 per cent ultimately died. Those figures, however, do not reveal the most interesting observation, namely, that of the patients subjected to simple drainage, only 35.5 per cent had an immediately favorable outcome, whereas 38.7 per cent required further surgery—some for the closure of persistent fistulas, but many to combat uncontrolled suppuration. On the other hand, of the patients subjected to radical drainage as an initial surgical measure, 60 per cent had an immediately favorable outcome and only 15 per cent required further surgery.

DR. JOHN ALEXANDER.—We should, I feel, stop discussing the medical treatment as distinct from the surgical treatment of pulmonary abscess. In the great majority of cases the two forms of treatment should be closely associated.

Acute and subacute abscesses should, I believe, be treated by absolute bed rest, postural drainage every one or two hours during the patient's waking hours, shrinkage of the swollen bronchial mucosa by the bronchoscopic application of equal parts of 1:1,000 adrenalin and 10 per cent cocaine approximately every five days, and temporary interruption of the phrenic nerve in order to quiet and relax the inflamed and cavernous portion of the lung. If, in spite of such a combination of measures, the abscess should fail to become smaller, direct surgical drainage should be considered.

May I express a word of warning against the use of a tube for the drainage of an abscess? Even a soft tube increases the danger of hemorrhage from pressure necrosis of a blood vessel exposed in the wall of the abscess. Furthermore, an open tube establishes an airway between the open bronchi in the wall of the abscess and the surface of the body so that the force of coughing is in part dispelled rather than entirely utilized in driving secretions toward the mouth. Since, therefore, the pulmonary secretions are not efficiently expelled through either the tube or the mouth, some of them may remain in the lung and be driven to hitherto uninfected portions of the lung, infecting them. The surgical removal of the external wall of the abscess and the snug packing of the cavity with moist gauze so as to occlude the mouths of the bronchi, tend to establish an efficient mechanism for expectoration and to remove one of the greatest dangers inherent in the surgical drainage of an abscess.

DR. RALPH B. BETTMAN.—Dr. Cutler made the remark that the location of the abscess had little to do with whether or not the patients got well without operation. By location he referred to the lobe in which the abscess was situated. From my own personal experience, I should say that the location of the abscess played a very important rôle in the healing, if the location were estimated not according to the lobe the abscess happened to be situated in, but according to its relation to the hilus or the periphery of the lung. It has seemed to me that those abscesses situated

in the periphery are much less likely to drain spontaneously than those near the hilus. I would therefore like to ask a favor of Dr. Cutler—a favor which because of the remarkable way in which he has followed up this series of cases should be easy to grant and might lead to some interesting results, and that is this: I wonder whether he could check over his series again and see whether there is or there is not any correlation in the frequency of spontaneous drainage if the abscess is situated near the hilus or near the periphery.

About ten years ago I did some work on dogs and found that the best way to obtain firm adhesions was to put a foreign body between the pleural layers and leave it until firm adhesions had developed. Adhesions so formed clinically have proved to be satisfactory and strong, and I wonder whether it is not safer to insert a pack in the pleural space over the site of abscess than to stitch the lung to the chest wall, as Dr. Cutler suggested. I cannot help feeling that there is a certain element of danger from infection, hemorrhage and spontaneous pneumothorax in the suturing of these pathologic lungs.

On account of the work of Pilot and Davis at the Cook County Hospital, Chicago, the use of arsphenamine has been almost routine in lung abscess; and, although I have no available statistics, I have the feeling that this form of treatment has been disappointing.

DR. EMILE HOLMAN.—Dr. Graham has given us a picture of lung abscess simulating tumor. We have recently undertaken the study of eight cases of carcinoma of the lung, simulating pulmonary suppuration. You are all familiar with the experience of mistaking inflammatory processes in the abdomen for carcinoma, and the same is certainly true in the chest. The clinical diagnosis of pulmonary suppuration in our cases was made, of course, with the aid of roentgenograms and of laboratory examinations, but at autopsy the lesion was found to be upon the basis of a primary carcinoma of the lung.

DR. ELLIOTT C. CUTLER.—I felt relieved that Dr. Allen's mortality was about the same as ours, because one always feels apprehensive in presenting such a high mortality rate. But the facts are there, and the facts are in the literature, as has been brought out in the discussion already. Dr. Alexander and Dr. Lilienthal pointed out the same thing when they said that the matter of medical and surgical treatment must be separated. I cannot agree, however, that one can say in any given case that this case should have medical treatment and this next patient should have surgical treatment. I believe that each case should have the advantage of at least some preliminary medical treatment before surgery is attempted, because there are many patients who have early abscess of the lung who are successfully treated by this method.

I do not know what a "progressive" abscess is, but I would treat abscess of the lung exactly as I would treat a pyloric ulcer in the stomach. If the abscess persists, one should watch it and give medical therapy for six weeks. If under those circumstances it fails to improve, the surgeon should operate. We know that most ulcers of the stomach can be cured very well with medical therapy, but some cannot. In the early phase, abscess of the lung is a pneumonitis. Nature has not walled it off. Which case will improve with postural drainage, sanatorial treatment and adequate care one cannot foretell, but certainly some will do so.

The matter of separating spirochetal gangrene from abscess of the lung as pathologic entities has been for a long time argued. Dr. Klein of Cleveland, to whom great credit should be given, feels that there is a definite form of pneumonitis due to the mouth organisms, which forms a typical picture, and which is quite separate from abscess. There may be, of course, secondary invasion of an abscess with spirochetes and other mouth organisms. Experimentally, we have produced abscess

of the lung with simple septic organisms and found such abscesses healed promptly. If spirochetes were added to the abscess, it became chronic. Clinical experience reveals that the chronic abscesses have additional contamination with mouth organisms. With gangrene of the lung we generally see a fulminating condition that goes on to death within two or three weeks, and at autopsy the whole lung is usually found to be involved.

I do not know the answer to Dr. Butler's question except that there is lower mortality rate with adequate drainage. I have long felt that this was assisted by using a vertical incision. This permits removal of ribs above or below and seems to diminish the number of terrible skin infections that sometimes creep out into the thoracic wall itself and result in gangrene of the skin and chest wall.

The last lesson about abscess, I think, is that it is very difficult to give these people adequate after-care in a general city hospital. The nurses are not keyed up to it and the patients need sanatorial after-care. I have among the cases studied for this presentation a patient, apparently cured for two years, who came back with recurrent abscess and died of it. We must study our cases over five, ten or fifteen year periods to know whether they are really well. Seeing patients only at the end of six months does not allow us to report them as well, for several will return later with recurrent abscess.

DR. C. I. ALLEN.—I have very little to add. The tube drainage was used only in those cases that had a complicating empyema. The statistics were drawn from cases over a considerable period of time. The conclusions, however, I believe, are warranted from the results which have been obtained in the later group of cases, in which we have been able to reduce the mortality rate considerably. Then in the two-stage operative cases, after eliminating the carcinoma cases, the mortality rate is lower than that for the group as a whole.

Of course statistics can be made to say almost anything, but I believe one is justified in drawing certain conclusions from one's own series and from the experience as expressed by others in the literature.

DR. HAROLD NEUHOF.—May I make some remarks concerning abscess of the lung rather than about the subject of mediastinitis which I presented. I do not think it is fair for us to go away with the feeling that all agree that the picture of abscess of the lung is one which has been portrayed this afternoon. I should like to refer to the fact that there are other viewpoints in connection with the surgical treatment of abscess of the lung. My own, for example, consists essentially in the idea that if the patient is not doing satisfactorily, even in the acute stage, it is quite proper to consider operation under such circumstances and not to await the tragic sequelae of the chronic stage of the disease.

I shall not burden you with details at this late hour, but I should like to refer for a moment to thirty-nine consecutive patients who have been operated upon for abscess of the lung in the acute stage, namely, within six weeks from the onset of pulmonary symptoms. Not all patients who were seen with acute abscess of the lung were operated upon. Only those who seemed to do unsatisfactorily under the forms of treatment which have been referred to this afternoon were subjected to operation. Undoubtedly, among them a certain proportion might have gotten well spontaneously, as indicated by Dr. Cutler and many others. Specifically in rebuttal to Dr. Cutler's comments I should like to point out the characteristics of the abscess of the lung as seen at operation in the acute stage. First, the abscess was an abscess as such, even when operation was performed as early as two weeks from its onset. Second, the abscess was always situated superficially in the lung, close to the parietal pleura and was a localized lesion, not a diffuse pneumonic process. Third, there were always overlying pleural adhesions, usually to the parietes, or in some instances between lobes or between lower lobe and diaphragm.

The picture of diffuse pneumonitis, which has been referred to today as a reason for not considering the question of operation in the acute stage, must be not only unusual but also of no great clinical import. I have not encountered it at operation in any of my cases. Of the thirty-nine patients operated upon for acute abscess of the lung thirty-eight are well. And when I say "well" I mean entirely well over periods up to as long as ten years. One patient died. I think that today, with better methods of localization, that patient would not have died because the operative approach was not correct.

Perhaps there will be some mortality in future cases, and it may be that the viewpoint I hold on operation for abscess of the lung in the acute stage is not correct, but it certainly seems unwarranted, when one is presented with the sad statistics we heard this afternoon, to accept the plan of treatment which was offered as the best plan for the management of the problem of putrid abscess of the lung.

ETIOLOGIC FACTORS IN THE PATHOGENESIS OF PUTRID ABSCESS OF THE LUNG*

LEO STERN, D.D.S., F.A.C.D.
NEW YORK, N. Y.

THERE is nothing essentially new in the concept that aspiratory diseases of the lungs may often be induced by inhalation of infected secretions from the mouth. The recent literature of medicine and dentistry is replete with clinical observations based on this hypothesis. More specifically, Franken,[1] stressing the need for preoperative dental hygiene, assumed a causal relationship between oral sepsis and postoperative pneumonia, a view later supported from the bacteriologic viewpoint by Appleton.[2] Pilot, Davis and Shapiro[3] believed that a definite connection exists between pyorrhea alveolaris and secondary infections of the lung in pulmonary tuberculosis. In a study of putrid infections of the lungs complicating silicosis, Proske and Sayers[4] placed great emphasis on finding that most of their patients presented neglected, diseased teeth and gums. Noting that a fairly characteristic combination of fusospirochetal organisms seemed to contribute to the complaint, these investigators presumed that the symptoms of putrid bronchial and pulmonary disease in silicosis result from direct aspiration of the mouth flora, mixed with fine dustlike particles of hard rock. They supposed that these particles induce a catarrhal inflammation, thus preparing the soil for the activity of oral anaerobes which are avirulent to the lung tissue.

Kline and Berger[5] linked up "Miller-Vincent" infections of the gums with pulmonary abscess and pulmonary gangrene, stressing the hazard attending the aspiration of infected material from the mouth during general anesthesia. Neuhof and Wessler[6] and Touroff and Moolten[7] strongly emphasized the fact that aspiration of infected particulate material from the mouth and throat is the usual mechanism in the production of postoperative putrid abscess of the lungs. The alternative theory of embolic transmission receives scant support.

It has been shown by many clinical investigators[8, 9, 10] that material from the mouth is readily aspirated under both local and general anesthesia. Lowenthal[11] produced an interesting sidelight to this view by demonstrating that buccal secretions invariably find their way into the tracheobronchial tree during general anesthesia administered for operations upon regions entirely unconnected with the oral or nasal cavities.

*From the Medical Services and the Surgical Service of Dr. Neuhof, Mount Sinai Hospital.

Received for publication Aug. 3, 1936.

In a recent communication I[12] directed attention to the high incidence of pulmonary abscesses following operations in the mouth, many of them performed under local anesthesia, with no evidence in the cases cited, of gross aspiration of teeth or fillings. It was assumed that the initial mechanism producing the abscess of the lung in many of these cases was the inhalation, not of organisms or secretions alone, but of minute particles of septic material, *particularly dental tartar,* which had become dislodged from the teeth during operation. A dulled or absent cough reflex eliminated the usual physiologic hindrance to the passage of this material into the bronchi. Indeed one case history was presented (Case 12) in which symptoms of pulmonary abscess developed within the usual incubation period, following simply the prophylactic removal of tartar from the teeth, and in which no anesthetic was employed. It was suggested that in this case particles of loosened dental tartar were aspirated, possibly during sleep.

ETIOLOGY OF PULMONARY ABSCESS

The present report deals with observations and conclusions regarding the etiology of putrid abscess of the lung in a series of 115 unselected, consecutive cases studied at the Mount Sinai Hospital.

A survey of the large number of well-authenticated case histories of *postoperative* pulmonary abscess available in this group verified the accuracy of the observation made by Neuhof and Wessler (loc. cit.) that the usual period for the development of these infections is within two weeks after operation. Accordingly in each case the search for etiologic factors focussed on the two-week interval immediately preceding the onset of symptoms.

In this series *no* known etiologic factor could be elicited in 64 per cent of cases notwithstanding a most searching inquiry in this direction. (Table I.) This percentage of cases of unknown etiology, it may be noted, is substantially identical with the figure quoted in my previous report based on 70 cases. *Findings of teeth heavily encrusted with tartar associated with markedly putrescent gums in the mouths of a large proportion of patients in this group, strongly suggested the likelihood that aspiration of material from the teeth was the cause of putrid pulmonary abscess in cases without obvious etiology.*

TABLE I

GROUPING OF CASES ACCORDING TO ETIOLOGY

ETIOLOGY	NUMBER OF CASES	PERCENTAGE
Posttonsillectomy	18	16.0
Postoperative (dental)	13	11.3
Postoperative (other types)	4	3.5
Postsubmersion	1	0.9
Postepileptic seizure	3	2.6
Postalcoholic stupor	2	1.7
Unknown	74	64.4

MECHANISM OF ASPIRATION

If it is true that aspiration of septic particulate material into the bronchi produces abscess of the lung, it is almost axiomatic to assume that this phenomenon generally occurs at times when the reflex mechanism governing the expulsion of foreign bodies from the throat, namely, the gagging and cough reflexes, is dulled or is absent. Among the pathologic states that may diminish the activity of these reflexes are episodes of unconsciousness due to drugs, injuries, or disease. The recurrent physiologic phenomenon that produces this situation is sleep. One may observe, moreover, that the heavy snoring of deep sleep is associated not only with a wide amplitude and rapid vibration of the soft palate, uvula, fauces, and tonsils, but not infrequently is accompanied by violent inspiratory efforts which may suck material out of the oral cavity and into the lungs.

If the tonsils are enlarged and spongy, this agitation may dislodge infected plugs from their crypts. These may then be readily aspirated. A degree of clinical support of this possibility was found in two cases of this series listed among the group of unknown etiology (Cases 27, 28, Table II) in which the initial signs of pulmonary abscess developed exactly two weeks after attacks of "acute tonsillitis" and in which no other etiologic factor could be elicited. Although it is conceded that such an occasional observation does not constitute proof, it seems sufficiently suggestive to warrant noting the possible etiologic significance of acute throat infection within the recognized incubation period.

If abscess of the lung is usually aspiratory in origin and the premise is correct that inhalation of loose particles in the mouth may occur readily during sleep, little further argument is required to develop the conclusion that dental sepsis may at times bear an etiologic relationship to this disease. Ideal material is available in unhygienic mouths in the form of friable and easily dislodged fragments of dental tartar bearing the anaerobes commonly identified with these infections of the lung. Material admixed with oral secretions is readily aspirated, especially during unconscious states. Fortunately more than merely the invasion of the lung tissues by this material seems necessary to produce an abscess. However, if the aspirated material lodges in a small caliber twig of the bronchial tree, the initial pathologic stage of pulmonary abscess is set up. (Touroff and Moolton, loc. cit.) These assumptions are supported in the laboratory by the work of Harkavy,[13] Smith,[14] Joannides,[15] Crowe and Scarf,[16] and Cohen,[17] who found that experimental production of abscess of the lung in animals could be induced if both foreign material and anaerobes of the fusospirochetal group were implanted in the tracheobronchial tree. I believe that ample *clinical* corroboration of this hypothesis has been found in the group of cases of pulmonary abscess of unknown etiology that forms the basis of this report.

METHOD OF STUDY

The mouths of this series of 115 patients were systematically examined with a view to determine their hygienic status. Not a few, as was to be anticipated in any group of unselected ward patients, showed marked oral sepsis. The striking fact that became evident as the investigation proceeded, however, was the overwhelmingly large number of patients displaying teeth and gums of an unhygienic, septic appearance in the group for which no known etiologic factor could be determined.

The mouths in these cases presented a fairly uniform picture. Large masses of tartar were found at the necks of the teeth, especially on the inner surfaces of the badly stained lower anterior teeth. This was generally of a light yellow color and of a soft, chalky consistency, permitting fragments to chip off quite readily. Deposits found about the necks of the remaining teeth were far less abundant, darker, usually brownish green in color and considerably harder. The gums were inflamed, and of a bluish color at the gingival margins. They were more or less receded, everted, and bled quite readily. Pressure applied just below the gingival margin resulted in the extrusion of purulent material.

Flakes of tartar dislodged for purposes of examination had a foul odor which often persisted after thorough washing with water. The organisms recovered by culture from this material varied, covering a large range of usual oral flora, but smears were generally marked by profusion of spirilli and fusiform bacilli. In addition to streptococci, anaerobic culture has yielded *B. melanogenicum*, *B. fusiformis* and *T. microdentium*, organisms frequently recovered at operation from pus in abscess of the lung. (Cohen.[18]) It might be supposed that this dental symptomatology results from the expectoration of foul sputum that was abundantly present in many cases. Such reasoning, however, fails to take into consideration the fact that many individuals not suffering from pulmonary disease may present an oral picture of this kind. Such cases are often designated ulcerative gingivitis or Vincent's gingivitis in dental literature and are reported to yield bacteriologic findings similar to those mentioned above. Smith[19] assumed a relationship between Vincent's gingivitis and abscess of the lung on the grounds of the identity of bacteriologic findings. A further and conclusive piece of evidence which quite absolves foul expectoration as the cause of these oral conditions is the discovery of the high percentage of clean teeth found in the group of putrid pulmonary abscesses occurring after tonsillectomy indicated in Table II. This point will be elaborated in the section to follow.

For purposes of this study an arbitrary classification of varying degrees of sepsis surrounding the teeth, graded one to four plus, was

selected—+ served to indicate a high degree of mouth cleanliness, complete absence of tartar, and firm, pink gums. The typical neglected, septic mouth described above was designated ++++.

Intermediate ratings denoted a varying range in amount of tartar deposit and gingival inflammation. The mouths classed +++ showed marked hyperemia of the gums and large accumulations of dental calculus, but the evidences of purulent discharge were scarcely as pronounced as in the ++++ rating; many in this group showed a large number of missing teeth. In Table II the one- and two-plus ratings are classified as nonseptic; the three- and four-plus ratings as septic.

TABLE II

ETIOLOGY	NONSEPTIC		SEPTIC		PERCENTAGE OF SEPTIC TEETH AND GUMS
	+	++	+++	++++	
Posttonsillectomy	15	1	2	–	11
Postoperative (dental)	1	2	3	7	77
Postoperative (other types)	2	1	1	–	25
Postsubmersion	1*	–	–	–	0
Postepileptic seizure	1	1	–	1	33
Postalcoholic stupor	2	–	–	–	0
Unknown	6†	6‡	32	30	84

*Patient was edentulous.

†Three patients were edentulous.

‡Includes 2 patients with history of tonsillitis immediately preceding abscess of lung.

As indicated in Table II, the patients with pulmonary abscess of unknown etiology showed a strikingly high percentage of septic teeth and gums.

Gingivitis associated with massive deposits of tartar is a disease that becomes more prevalent from the third decade of life onward and is more frequently observed in the male. That the irritating effects of dental tartar constitute the chief cause of chronic gingivitis is a generally accepted fact and is supported by the evidence that the gum inflammation often resolves only after the tartar is removed. The small percentage of mouths that could be classified as septic in the posttonsillectomy group may be partially explained by the fact that the average age of this group was quite low, including a number of children, and predominantly (81 per cent) female. Conversely there is an absence of "unknown" etiology cases among the children in this series, the percentage of such cases distinctly rising with age. (Table III.) It is interesting to note that in the group of unknown etiology, showing a remarkably high incidence of unhygienic teeth and gums, there was a preponderance of males (72 per cent) and that most of the patients were over thirty years old. In view of the fact, however, that as many as 84 per cent of patients in the group of unknown etiology disclosed a marked degree of clinical dental sepsis (classified +++ and ++++) it seems fair to assume that a relationship exists between these dental conditions and abscess of the lung that may reasonably be considered

to have an etiologic significance. This assumption is fortified by the similarity of dental findings in the group representing pulmonary abscesses occurring as a sequel to dental operations, the mechanism of which has already been described.[12] As this mechanism was assumed to depend largely on the aspiration of infective particulate material, it is significant that this is the only other group showing a comparably high percentage of tartar encrusted teeth.

TABLE III

RELATIONSHIP OF AGE TO INCIDENCE OF DENTAL SEPSIS

AGE GROUP	TOTAL NO. OF CASES	CASES SHOWING DENTAL SEPSIS	PERCENTAGE
1-20 years	9	0	0
21-30 years	12	2	16.7
31-40 years	37*	26	70.0
41-50 years	44*	38	86.0
51-70 years	13†	9	69.0

*One patient was edentulous.
†Two patients were edentulous.

TABLE IV

AGE GROUPS IN GROUP OF UNKNOWN ETIOLOGY

AGE GROUP	TOTAL NO. OF CASES	NO. OF CASES IN UNKNOWN ETIOLOGY GROUP	PERCENTAGE
1-20 years	9	0	0
21-30 years	12	2	16
31-40 years	37*	24	65
41-50 years	44*	41	93
51-70 years	13†	6	46

*One patient was edentulous.
†Two patients were edentulous.

The number of cases of dental and gingival pathology of the type described increased decidedly with succeeding age groups. There was a like increase in the incidence of putrid pulmonary abscess, particularly of the unknown etiology type, with advancing age. All putrid abscesses of the lung found in children in this series occurred after tonsillectomy. The teeth in such cases were free from tartar and the gums were healthy.

CONTROL OBSERVATIONS

In order to decide whether this marked preponderance of septic, tartar encrusted teeth found in the unknown etiology group could be accounted for purely on the basis of such factors as sex, age group, lowered resistance due to disease, or social condition, two controls were employed.

The first consisted of 100 consecutive unselected patients referred from the medical wards of the hospital for dental consultations. This group consisted of chronically and subacutely ill patients most of whom

were examined for dental sources of focal infection. The average social and economic status of these patients may be assumed to be substantially the same as that of the pulmonary abscess group. Sixty-seven per cent of this number were males; the average age of the group was thirty-six years. Periapical infection disclosed by roentgen ray examination was eliminated from consideration, only clinical findings of "septic" gums and unhygienic teeth being noted. Nine cases were classed +++, 3 cases ++++. This figure of 12 per cent of gingivitis associated with heavy tartar deposits probably represents fairly accurately the average of such conditions for patients in the wards of the Mount Sinai Hospital.

The second control comprised 50 consecutive unselected patients presenting themselves for a check-up in the chest follow-up clinic. Most of these patients were suffering from recent or active manifestations of chronic pulmonary diseases, other than abscess of the lung, in the following variety and number: 14 bronchiectasis, some associated with pneumonitis; 7 malignant and 6 benign neoplasms of the lungs or bronchi; 3 pulmonary tuberculosis; 3 empyema; 2 atelectasis; 2 lobar and 2 postoperative pneumonia; 2 emphysema of lungs; and one of each of subphrenic abscess, syphilitic aortitis, gumma of auricle, nodular stenosis of bronchus, leptothrix infection of lungs, bronchial bleeding in polycythemia, aneurysm of the pulmonary artery, pulmonary fibrosis, broncholith.

This group likewise consisted of a large majority of males past the third decade of life. Four members of this group were classified +++, three as ++++, a percentage of 14 demonstrating marked dental sepsis. It can therefore be concluded that pulmonary disease per se is not associated with an unusually high percentage of septic mouths.

As previously suggested, an objection which might be raised to the theory I have advanced is that the preponderance of cases of septic gums discovered in the group classified as of unknown etiology was an effect rather than the cause of the pulmonary abscess. This point is particularly relevant in view of the fact that many of these patients expectorated quantities of pus. It could be urged that this putrid material intermittently spread over the teeth might lead to the pathologic changes about the teeth. However, a study of the oral findings in the posttonsillectomy group in which the amount, character and duration of the expectoration were substantially identical with that of the cases of unknown etiology refutes such an assumption. Indeed the percentage of remarkably clean, healthy mouths in this group was unusually high and demonstrates conclusively that expectoration of putrid material is in itself not responsible for heavy tartar deposits or gingivitis.

In view of the contention that these mouths presenting massive tartar deposits and suppurative gingival infection are linked up etiologically

with idiopathic cases of pulmonary abscess, the question naturally arises: why do not oral lesions of this type result in abscess of the lung more frequently? The answer to this must be that the pathologic lesion in the lung is the result of a series of so many coincidences that the mathematical chance of their occurrence is small. Also many a small abscess may produce such mild symptoms that the case is not seen by the physician. The seed and the soil must be favorable at almost the same moment. It may be said with reasonable conservatism that the seed in these mouths becomes available at those infrequent moments that tartar is dislodged and floats loosely in the oral secretions. Favorable opportunities for its free passage into the tracheobronchial tree occur only when the cough reflexes are inactive. Furthermore this material must reach and become fixed in a more or less peripheral portion of the bronchial tree. Finally, sealing must take place in order that anaerobic growth may continue without interruption. It is also possible that a small amount of blood such as by coagulation may serve to seal the small bronchus, is necessary. In this connection one may add that recurrent bleeding is common in these pyorrhetic mouths.

CLINICAL CORROBORATION

Possibly the most conclusive clinical indication that could be presented in support of the theory expounded would be to demonstrate that more than one abscess of the lung may occur in a patient with pronounced gingival disease and in the absence of any other recognized etiologic factor. The following case report, number 36 in the "unknown etiology" group, is offered in evidence:[20]

Hospital No. 371988. J. M., a forty-two-year-old workman, was admitted to the hospital in October, 1934. He had previously been a patient at the Mount Sinai Hospital in October, 1922, suffering from an abscess of the lung in the right upper lobe for which no satisfactory etiologic factor could be elicited. At that time the teeth were encrusted with tartar and the gums were congested and suppurative. Pneumonotomy and drainage resulted in cure of the pulmonary abscess.

The interval history was uneventful until the present illness began. Three and one-half weeks before admission a mild cough with white mucoid expectoration developed. Ten days later the patient experienced a violent chill, lasting twenty minutes, followed by high fever, a more severe cough and expectoration of foul smelling sputum. Severe pain in the left lower chest anteriorly occurred a few days later, with hemoptysis.

The mouth was found to be markedly unhygienic, with large deposits of flaky tartar particularly evident about the lower anterior teeth. The gums were inflamed, and pressure at the gingival margins of the anterior and upper posterior teeth was followed by extrusion of pus. The patient volunteered the information that as far back as he could remember he had suffered episodes of "sore mouth with profuse bleeding of the gums" recurring at approximately six-month intervals. The last episode of this sort had occurred three months before, at which time a maxillary right molar was removed under local anesthesia.

Clinical, bronchoscopic, and x-ray studies resulted in a diagnosis of acute abscess of the lung in the lower left lobe, with marked pleuritis. This was confirmed at operation.

COMMENT

This patient had been discharged as cured of an abscess of the lung in the right upper lobe nearly eleven years ago. A second acute abscess of the lung occurred recently, situated in the left lower lobe. Studies in the hospital failed to indicate any relationship between the two lesions.

An interesting angle is presented from the standpoint of pathogenesis. The history revealed no recognized etiologic factor in either instance. It is logical to assume that the same cause operated to produce the result on each occasion.

In searching for a common factor that could have been reasonable for the production of both these lesions, attention is directed to the condition of the teeth and gums. If aspiration of septic particulate material is the accepted etiologic factor in most abscesses of the lung, and if aspiration occurs readily from the mouth, there was ample suitable material in this case to furnish factors for the production of each pulmonary abscess. Because of the unusual nature of this case and because of conclusions which had already been crystallized in a study of many cases of pulmonary abscess of unknown etiology, a most searching examination of the history was made. No other causative factor could be elicited. It is presumed that in this case some of the small fragments of tartar that were dislodged from time to time were aspirated, and on two widely separated occasions produced a putrid abscess of the lung.

SUMMARY AND CONCLUSION

In 64 per cent of pulmonary abscesses in the present series, comprising 115 cases, no known etiologic factor was disclosed. The mouths in 84 per cent of this unknown etiology group presented a typical and uniform picture of marked dental disease, characterized by large masses of tartar deposit and infected gums. In view of this high incidence and in view of the fact that the infected material adherent to the teeth in these cases is readily dislodged and may be aspirated during sleep, it is believed that a reasonable explanation of the pathogenesis of these cases has been offered. Reasons were given for the belief that the expectoration of foul sputum does not adequately account for the dental lesions. Control observations indicated that neither age, sex, illness, nor social status could account for the high percentage of oral pathology. These characteristic lesions of the gums were disproportionately common only in patients with putrid abscess of the lungs in whom the etiology of the pulmonary abscess was unknown.

REFERENCES

1. Franken, S. W. A.: Prophylaxis of Postoperative Pneumonia by Oral Hygiene, Arch. Int. Med. 48: 1225, 1931.
2. Appleton, J. L. T., Jr.: Bacterial Infection With Special Reference to Dental Practise, Philadelphia, 1933, p. 401, Lea & Febiger.

3. Pilot, I., Davis, D. J., and Shapiro: Studies on Fusiform Bacilli and Spirochetes. VI. Their Association With Pulmonary Tuberculosis in Cavities, Bronchiectasis and Gangrene, Am. Rev. Tuberc. **8**: 249, 1923.
4. Proske, H. O., and Sayers, R. R.: Pulmonary Infection in Pneumoconiosis, U. S. Public Health Reports **49**: 839, 1934.
5. Kline, B. S., and Berger, S. S.: Pulmonary Abscess and Pulmonary Gangrene: Analysis of Ninety Cases Observed in Ten Years, Arch. Int. Med. **56**: 753, 1935.
6. Neuhof, Harold, and Wessler, H.: Putrid Lung Abscess: Its Etiology, Pathology, Clinical Manifestations, Diagnosis and Treatment, J. THORACIC SURG. **1**: 637, 1932.
7. Touroff, A. W. S., and Moolten, S. E.: The Symptomatology of Putrid Abscess of the Lung, J. THORACIC SURG. **4**: 558, 1935.
8. Myerson, M. C.: Bronchoscopic Observations of the Cough Reflex in Tonsillectomy Under General Anesthesia, Laryngoscope **34**: 63, 1924.
9. Iglauer, S.: Aspiration of Blood Into the Larynx and Trachea During Tonsillectomy Under Local Anesthesia, Ann. Rhin. & Laryng. **37**: 231, 1928.
10. Ochsner, A., and Nesbit, W.: Bronchography: Introduction of Iodized Oil Into the Tracheobronchial Tree by the "Passive" Method, Am. J. M. Sc. **175**: 175, 1928.
11. Lowenthal, G.: Tracheobronchial Aspiration of Buccopharyngeal Secretion During Ether Anesthesia: Immediate Postoperative Bronchoscopic Study of 21 Patients, Arch. Otol. **21**: 561, 1935.
12. Stern, L.: Putrid Abscess of the Lung Following Dental Operations, J. THORACIC SURG. **4**: 547, 1935.
13. Harkavy, J.: The Pathogenesis of Aspiratory Abscess of the Lung, Arch. Int. Med. **43**: 767, 1929.
14. Smith, D. T.: Experimental Aspiratory Abscess, Arch. Surg. **14**: 231, 1927.
15. Joannides, M.: The Etiology of Pulmonary Abscess, Surg. Gynec. Obst. **47**: 449, 1928.
16. Crowe, S. J., and Scarf, J. E.: Experimental Abscess of the Lung in Dogs, Arch. Surg. **16**: 176, 1928.
17. Cohen, J.: Personal communication.
18. Cohen, J.: Bacteriology of Abscess of the Lung and Methods for Its Study, Arch. Surg. **24**: 171, 1932.
19. Smith, D. T.: Oral Spirochetes and Related Organisms in Fuso-Spirochetal Disease, pp. 118-120, Baltimore, 1932, Williams & Wilkins Co.
20. Stern, L.: Two Pulmonary Abscesses in a Patient, With a Fourteen-Year Interval, J. Mt. Sinai Hosp. **3**: 118, 1936.

745 FIFTH AVENUE

ABSCESS OF THE MEDIASTINUM*

ROBERT C. FISHER, M.D.
NEW YORK, N. Y.

HARE[1] collected 115 cases of mediastinal abscess in 1889. Two-thirds of them were instances of acute abscess, and the great majority occurred in males. The anterior mediastinum, that is, the space between the sternum in front and the pericardium and heart behind, was commonly the site of the suppuration. Trauma of the chest was the most frequent cause, but a localized collection of pus—an abscess—may form in the mediastinum, either anterior or posterior, as a result of infection of lymph glands in this region, or disease of the esophagus, trachea or main bronchi, or by extension of suppuration from the lung, pleura, pericardium, spine or sternum. Many cases, particularly the chronic abscesses, were of tuberculous origin. There is yet another way in which pus may collect in the posterior mediastinum. In the case to be presented, the abscess was located in the posterior mediastinum and occurred as a complication of a severe sore throat. Such an event is very rare following a nasopharyngitis, but during the 1918 epidemic of influenza a number of these cases were reported, and it is worth noting that the case under discussion showed the presence of *B. influenzae* both in the smear of sputum and in the blood stream. Furthermore it is exceptional for an abscess located in this situation to rupture spontaneously into a bronchus, drain adequately, and eventually heal.

CASE REPORT

This patient is a white female twenty-six years old, who was taken suddenly ill while at work in an office April 19, 1935. The illness started with a chill, followed by fever and a sore throat. She went home and to bed and was seen by her family doctor. The following day her throat continued sore so that she was unable to swallow. She vomited several times, and her temperature fluctuated between 100° and 101.6°. On the third day she began to cough, a dry hacking cough without sputum, and she was aware of a pain in the back high up between the shoulder blades. The throat continued painful, and her temperature rose about a degree higher. The next day her condition grew very much worse, with a jump in temperature to 105°. Her physician sent her to St. Vincent's Hospital for further observation. There was nothing of importance in her past, personal, or family history.

On admission she appeared acutely ill. Her throat was angry red; the tonsils were small, buried, and looked acutely inflamed; and the pharyngeal wall showed

*From the Medical Service of St. Vincent's Hospital.
Read before the Graduate Fortnight, New York Academy of Medicine.

swollen lymph follicles, and mucopus on its surface. No cervical lymph nodes were palpated. Temperature was 103.8°, pulse 128, and respiration 28. She presented the picture of a grip with acute tonsillitis and pharyngitis. A urine specimen contained a large amount of albumin, many casts and pus cells. A blood count showed 17,000 white blood cells, of which 81 per cent were polymorphonuclears and

Fig. 1. Fig. 2.

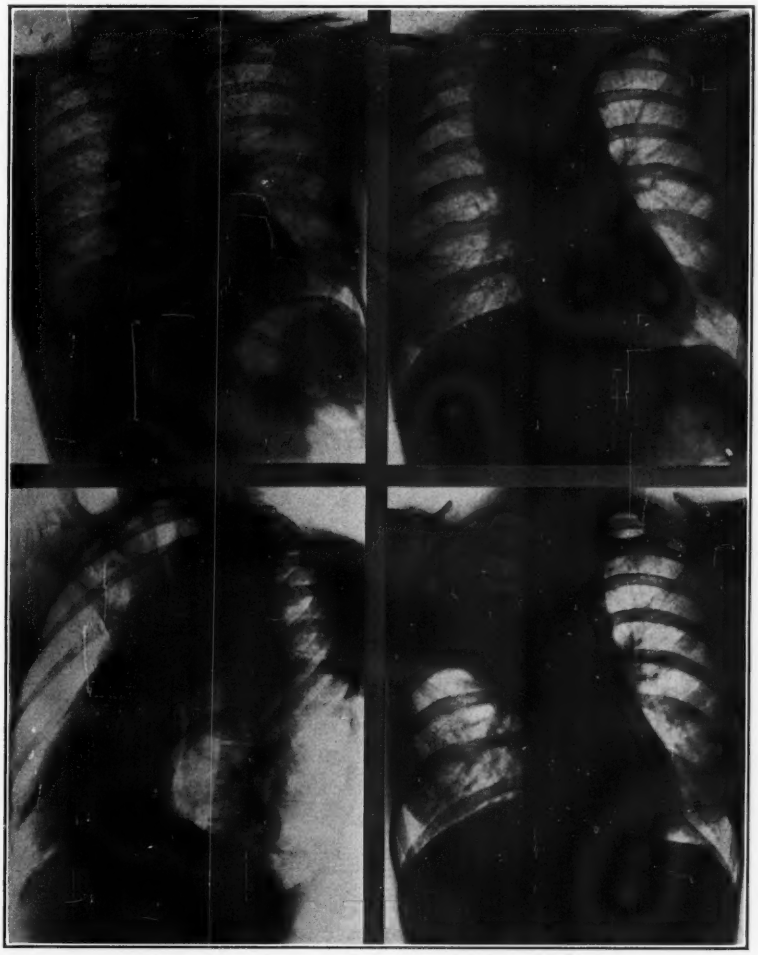

Fig. 3. Fig. 4.

19 per cent lymphocytes. During the first few days after admission, her throat continued to be painful, she had a nosebleed, her temperature stayed between 103° and 104° with the pulse curve at the same level, and she began to raise sputum, the character of which is not noted on the chart. Her appearance now was that of a patient with lobar pneumonia without definite signs of consolidation in the lungs. The sputum was examined and showed many influenza bacilli with a few

streptococci, but no pneumococci. A blood culture grew *B. influenzae.* This was repeated a few days later, and again *B. influenzae* was grown from the blood.

An x-ray film of the chest taken on April 26, the fourth day in hospital, showed an area of diminished ventilation along the vertebral aspect of the right upper lobe (Fig. 1). During the following few days the patient's temperature became somewhat irregular, fluctuating between 100° and 103°, and repeated physical examination of the chest failed to show any signs of consolidation. She was coughing,

Fig. 5.

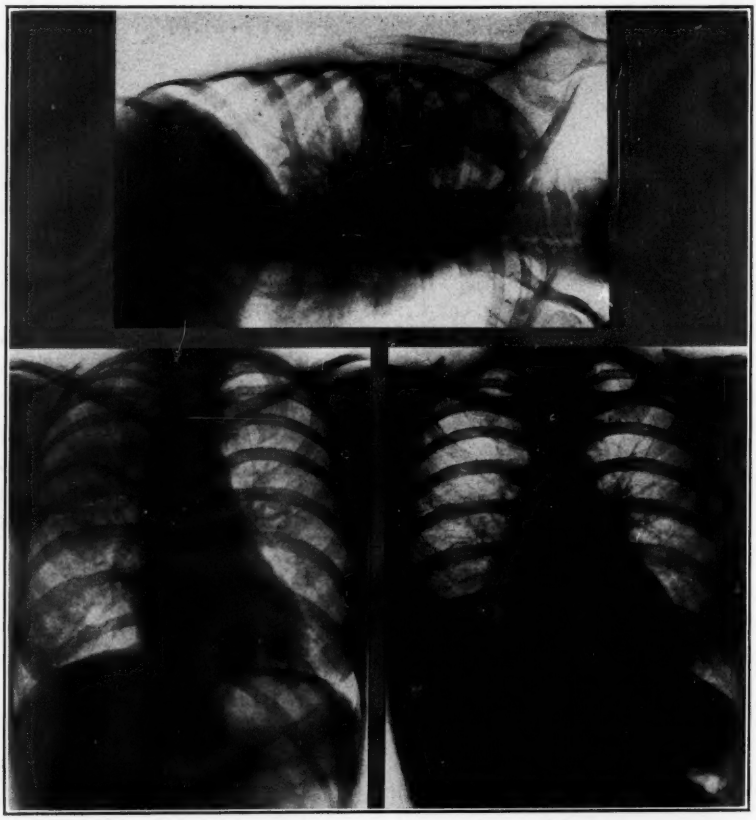

Fig. 6. Fig. 7.

but raising very little sputum. On May 2, the tenth day in the hospital, she was irrational and was transferred to the accident ward. The temperature now had dropped to between 100° and 101°, and another x-ray film taken May 1 was reported as follows: "The process has extended and its limits are now sharply defined on both sides of the spinal border, probably indicating that it is of mediastinal origin, and represents a glandular lesion or encapsulated mediastinal effusion." (Fig. 2.) A film taken in an oblique position showed the shadow to be in the posterior superior mediastinum (Fig. 3). At this time the patient complained of pain deep in the lower neck when swallowing and was tender to pressure on the trachea, behind the suprasternal notch, and to pressure and percussion in the right interscapular region

behind. Except for a few fine râles along the right sternal border in the second and third spaces, the lungs were clear, and the heart, abdomen and skin were negative. It was suggested at this time, two weeks after the onset of the sore throat, that the patient might have an abscess of the mediastinum, complicating a nasopharyngitis and tonsillitis.

For the next few days the patient's temperature again went up from 100° to 103°, and on May 5 she began to cough up small amounts of mucopurulent sputum. She could lie quietly in bed without dyspnea, and did not complain of any pain in the chest. Tenderness to pressure in the right interscapular region behind was still present. There was dullness to percussion above the third rib in front, and a few medium râles were heard in the second and third interspaces near the sternum. The temperature began to spike between 101° and 103°, the sputum gradually increasing in amount. It was coughed up in thick, pale greenish yellow clumps, and was mucopurulent and odorless. The patient complained of some difficulty in swallowing and at times was hoarse. On May 8 an x-ray film showed an extension of the mediastinal effusion into the right upper chest (Fig. 4).

From an examination of the x-ray films and clinical behavior of the patient up to this time, it is obvious that the abscess had extended from the posterior superior mediastinum into the upper lobe of the right lung, and had eroded a bronchus in the lung through which the pus was slowly being evacuated. A film of the patient lying on her left side confirmed this interpretation (Fig. 5). The physical signs at this time were diminished intensity of the breath and voice sounds, and a few coarse râles over the upper lobe. Slight dullness was also present. Over the lower lobe posteriorly and in the axilla, a few sibilant and sonorous râles were heard. Postural drainage was now started and the patient improved so that about ten days after the abscess had ruptured into a bronchus, her temperature had fallen below 100°. The sputum was the same in appearance, but during this time had increased in amount to between 2 and 3 ounces a day. X-ray examination showed a gradual reduction in the size of the cavity in the lung and mediastinum, and a resolution in the infiltration of the upper lobe of the lung (Fig. 6).

About three weeks after the abscess ruptured the sputum was reduced to less than an ounce a day, and its character had changed from clumps of pale greenish white mucopus to a thin serous material. During the following week the patient's temperature dropped to normal and her cough subsided. She was discharged well on June 15, eight weeks after admission. A chest film taken two months later showed complete resolution of the process (Fig. 7).

In 1924 Lerche[2] described the fascia and fascial spaces of the neck and thorax. The pretracheal fascia covers the trachea in front and blends laterally with the sheath of the vessels of the neck. The prevertebral fascia, covering the bodies of the vertebrae and muscles attached to the spine, also joins the sheath of the vessels in the neck laterally. Between this prevertebral fascia and the esophagus is a fascial plane of loose areolar tissue, continuous above with the buccopharyngeal fascia and below with the posterior mediastinum. This retroesophageal space extends from the base of the skull to the diaphragm, and is well seen at autopsy as one removes the trachea, esophagus and larynx en masse. Schmitt in 1893 injected the retroesophageal space through the base of the skull with moderate pressure and amounts of fluid, and found the injected fluid filled the space from

the base of the skull to the sixth thoracic vertebra. In the neck, the injected mass was behind the esophagus and extended laterally to the sheaths of the vessels. In the thorax, the esophagus was completely surrounded by the injection mass, and the lower end of the trachea and its bifurcation in the posterior mediastinum were likewise surrounded by the same material. We see, then, that infection and pus may spread from the retropharyngeal areolar tissue downward behind the esophagus into the posterior mediastinum, as in the case under discussion.

Lambert and Berry[3] reported a series of experiments in 1927, in which they attempted to ascertain the way in which a collection of pus in the posterior mediastinum might spread. Using dead infants from birth up to two years of age, they removed the spinous processes and laminae of the first three thoracic vertebrae and the corresponding part of the spinal cord. They then passed a needle on a syringe forward through the second thoracic intervertebral disc until the needle tip was in the posterior mediastinum, and injected a colored solution. By this method it was demonstrated that there are definite paths by which pus will spread from a focus in the mediastinum (1) through the areolar tissue in the root of the lung and the broad ligament to beneath the visceral pleura, and into the lung itself along, but outside the walls of the larger bronchi, and (2) posteriorly along the bodies of the vertebrae to the endothoracic fascia, here lying outside the parietal pleura, between it and the chest wall. The case under discussion demonstrates the first method of spread very well.

REFERENCES

1. Hare: Oxford System of Medicine, 1889.
2. Lerche: Arch. Surg., January, 1924.
3. Lambert and Berry: Arch. Surg., January, 1927.

DISCUSSION

DR. ADRIAN V. S. LAMBERT.—This case presents several features which are noteworthy and also directs our attention to the subject of treatment.

As Dr. Fisher points out, the anatomical fascial planes determine the route which cervical suppurations in the neck may take to reach the mediastinal space. When this condition occurs, a situation is present the seriousness of which is frequently not fully realized by the physician. In the present case the outcome was unusual and fortunate, as we rarely meet with a case of mediastinal abscess which heals spontaneously by rupturing into a bronchus. When this does occur, however, the point of rupture is usually situated on the posterior aspect of a main bronchus or the trachea, that portion which is devoid of cartilage. It is rare for the process to ulcerate through the visceral pleura and discharge through the alveoli. The usual course of such an abscess is not by rupture into a bronchus but to descend down into the posterior mediastinum to the diaphragm. It may extend laterally in the costovertebral gutter within the endothoracic space posterior to the parietal pleura. The process may indeed descend into the retro-

peritoneal space. In such a case the pus passes through the crus of the diaphragm via the openings for the passage of the great splanchnic nerves and not through either the esophageal or the aortic openings of the diaphragm. Should the process continue, the pus, lying as it does on the anterior surface of the quadratus lumborum muscle, may descend over the crest of the ilium and passing down on the surface of the iliacus muscle emerge beneath Poupart's ligament and "point" on the anterior surface of the thigh external to Scarpa's triangle. In no particular does it resemble a psoas abscess.

We believe that operative treatment or drainage of mediastinal abscess through a posterior mediastinotomy is the proper procedure, as it entails less risk than expectant treatment in the hope that it will rupture into a bronchus. With the experience of the last few years with extrapleural thoracoplasty the operation of posterior mediastinotomy is not so hazardous as most practitioners appear to consider it, but is a relatively safe procedure.

USE OF PHRENIC NEURECTOMY COMBINED WITH ARTIFICIAL PNEUMOPERITONEUM FOR COLLAPSE OF ADHERENT TUBERCULOUS LUNG

MINAS JOANNIDES, M.D., AND OTTO C. SCHLACK, M.D.
CHICAGO, ILL.

PLEURAL adhesions in many cases have been the cause of failure to collapse satisfactorily a tuberculous lung by pneumothorax. Some adhesions may be cut and in this way result in a satisfactory closure of cavities. In other cases the adhesions are so extensive that they necessitate the use of more radical procedures for the collapse of the lung. It is true that phrenic neurectomy alone in a large number of these cases has been of great assistance. Those in whom the cavities were too extensive or too rigid, or in whom the diaphragm did not rise satisfactorily, demanded more drastic procedures for satisfactory collapse. Of this latter group, many went on from bad to worse because they presented contraindications to thoracoplasty.

In this preliminary report we wish to propose a new principle in the treatment of patients in whom artificial pneumothorax is unsuccessful because of extensive adhesions. This principle involves the use of a preliminary paralysis of the hemidiaphragm on the diseased side and the introduction of air into the peritoneal cavity.

The modus operandi of this combination is as follows: (1) The paralyzed hemidiaphragm has a tendency to rise at inspiration. Because the paralyzed diaphragm remains relaxed and its suction power has been reduced to a minimum, the lung will tend to contract of its own elasticity and the surface tension of the liquid particles in the parenchymal structures. (2) The paralyzed hemidiaphragm will rise to a certain limit and remain there. In some cases the diaphragmatic paralysis alone is not sufficient to produce a tendency to cavity closure, and a mechanical pressure is necessary to compress the diseased lung. By injecting sterile, filtered air into the peritoneal cavity, and causing the patient to lie in a Fowler's position at an angle not greater than 45 degrees, we can introduce a sufficiently effective pneumatic splint to compress the diseased lung from the undersurface of the diaphragm. The effect of artificial pneumoperitoneum can be enhanced by the use of an abdominal support to increase the intraabdominal tension. (3) The healthy hemidiaphragm in contracting and relaxing intermittently acts as a pump and squeezes almost all the air over to the side of the paralyzed hemi-

From the Department of Surgery, College of Medicine, University of Illinois, Chicago; and the Cook County Tuberculosis Hospital, Oak Forest, Illinois.
Received for publication, September 18, 1936.

diaphragm so that little or no air remains underneath the nonparalyzed side. In this way a pneumatic pad can be maintained to collapse a larger portion of the lung. Because the air is absorbed much in the same way as in pneumothorax, refills have to be given until a satisfactory healing of the lung has resulted.

The technic requires the same armamentarium as in artificial pneumothorax. The point of injection should be at the left hypochondrium in order that the liver (a solid organ) be avoided in the course of the puncture of the abdominal wall. A sufficient amount of procain should be injected to anesthetize the tissues and, especially, the peritoneum. The patient should lie flat on the table or bed with the left side of the body at an angle of 45 degrees so as to allow for exposure of the left upper quadrant of the abdomen and also allow for the dropping of the underlying abdominal viscera to a position away from the abdominal wall. Thus, injury to the abdominal viscera may be minimized and possibly completely avoided. The needle should be of a short bevel type but sufficiently sharp so that excessive pain from pressure and undue trauma to the peritoneum is avoided. For the first injection 500 to 800 c.c. of air may be given and refills to be given in three to fourteen days, depending on the degree of rise produced. In the early cases the air is absorbed much more rapidly than in pneumothorax, thereby necessitating a closer observation of these cases. X-ray studies must be insisted upon rather than depending upon physical signs.

In the first two or three days after the introduction of air into the peritoneal cavity, the patients complain of pain in the shoulder of the nonparalyzed side and also of pain at a point in the infraclavicular area coinciding with the mammary line. They may also complain of tenderness and pressure in the abdomen. One patient stated that the pneumoperitoneum made her "feel hungry" all the time. Another patient complained of a gurgling sound in the abdomen and a tendency to belching. We had to remove 200 c.c. of air from a patient who complained of severe dyspnea after 800 c.c. of air was introduced. In another patient who received 800 c.c. of air and complained of tendency to dyspnea when the chest was elevated, sufficient absorption took place so that in twenty-four hours she was up and about and perfectly comfortable.

It is necessary to emphasize possible complications that result from artificial pneumoperitoneum, and particularly so because carelessness in the administration of air into the peritoneal cavity and faulty asepsis may result in serious complications and throw a useful measure into disrepute. After the idea was conceived by us, we hesitated for a long time in even attempting the procedure because of the thought that a complicating peritonitis might be fatal to the patient. When, however, we continued to see patients going from bad to worse and eventually dying we picked up enough courage to try the method and are getting satisfactory results. We always keep in mind the danger of perfora-

tion of part of the gastrointestinal tract. Similar arguments, however, being present against the use of artificial pneumothorax, and the relatively small number of complications have given us courage to use this method and propose it as a routine measure for patients in whom thoracoplasty may be contraindicated.

We are carrying on a series of clinical and experimental observations and hope in the near future to publish subsequent papers giving our results. Some of the problems under consideration are as follows: (1) The effect of artificial pneumoperitoneum on the gastrointestinal tract physiology, including motility and secretory activity. (2) The effect of artificial pneumoperitoneum on the peripheral circulation. (3) The effect of artificial pneumoperitoneum on urinary excretion and the blood content of nonprotein nitrogen, urea, and creatinin. (4) The effect of artificial pneumoperitoneum on respiration, including studies of the vital capacity, analysis of alveolar air, and the respiratory function of the blood. (5) The effect of bilateral phrenic neurectomy and artificial pneumoperitoneum. (6) The possible utilization of artificial oxypneumoperitoneum in the treatment of general peritonitis by preventing contact of infected material with the subphrenic area of the peritoneum that has the highest absorbing capacity, and by forcing the infected exudates toward the lesser absorbing pelvic area so that localization may occur. (7) The use of artificial pneumoperitoneum in tuberculous pregnant women who were allowed to go to full term. After the uterus is emptied, the artificial pneumoperitoneum may act as a balancer of intrapleural and intraabdominal pressures. By virtue of the slow absorption of the oxygen or air from the peritoneal cavity the circulation and respiration will have time to slowly readjust itself, and thus a fatal collapse may be prevented.

A thorough search of the literature which includes over 500 papers failed to show us that artificial pneumoperitoneum in combination with phrenic neurectomy has been previously used as a means of collapsing adherent tuberculous lungs. The papers heretofore published include the use of pneumoperitoneum as a diagnostic measure in perforation of the wall of the gastrointestinal tract, in gynecologic conditions and in spontaneous pneumoperitoneum complicating various types of abdominal pathology. It has also been used as a therapeutic measure in tuberculous enteritis and tuberculous peritonitis.

We hope that the term "artificial pneumoperitoneum" will be generally adopted for the therapeutic procedure to distinguish it from the spontaneous pneumoperitoneum, which is a pathologic state.

CHYLOTHORAX

Case Report

Oren A. Beatty, M.D.
Glasgow, Ky.

CHYLOTHORAX is an interesting symptom complex and mode of origin of chylous fluid may present a problem of diagnosis in both chest and abdomen. The case presented herewith has characteristics of unusual interest from the standpoint of history, of mode of origin of chylothorax and of possibilities for treatment.

J. I., sixty-three, a widow, was admitted to the chest clinic Oct. 17, 1935, with chief complaints of weakness, smothering, and gas on stomach. This smothering was aggravated by movement or leaning to left side or lying on left side. She also had complaints of "gurgling in the chest," and loss of weight. Patient states she was in an automobile accident June 17, 1928, and has not been well since. She states that the left clavicle was fractured at the junction of the inner and middle thirds with depression of the outer fragment. Following the accident she had pain in the left shoulder and in the precordial region. The precordial pain was constant but was also aggravated by respiration and persisted to some extent for two years. Immediately after the accident there was a small amount of hemorrhage from the nose and the mouth, and at intervals for six weeks frothy blood was expectorated after coughing. She was short of breath and had had wheezing the same length of time. Also following the accident there was engorgement of veins and swelling of the left arm and shoulder. The swelling of the arm subsided within three months, and the swelling of the shoulder subsided within one year. The veins of the left arm, shoulder, and breast were prominently engorged for six months with some subsidence after this time but with persistence to some extent to the present time. Accompanying the pains and the swelling of the left arm and shoulder was marked limitation of their use. Full use of this arm has not returned but has improved markedly.

Three years after the accident she had regained her former weight of 132 pounds. She was in good health except for pain in the left shoulder and the precordium, and limited use of the left arm and shoulder. She retained this status until April, 1934; during one night of that month she became suddenly nauseated, vomited, and had diarrhea for one day. Following this she lost weight rapidly and was at her lowest by July. There was no weakness and the appetite was fair. She regained to 129 pounds by January, 1935. She noted no unusual engorgement of the veins of the left arm and shoulder at that time.

In January, 1935, she had a severe "cold" with pain under the left scapula and less marked pain under the right scapula. She had a severe cough but with no expectoration. This "cold" was different from any other cold she had ever had in that she had "gurgling" in the right chest, wheezing in the right base, and shortness of breath with a feeling of fullness in the chest. This feeling of fullness was at its worst in June, 1935. The "cold" cleared up during July and August, but

Received for publication, July 16, 1936.

the other symptoms persisted until after admittance to the chest clinic. Accompanying the above symptoms were loss of weight, marked loss of strength, loss of appetite, and an aggravation of a tendency to flatulency.

The past history was essentially negative. From the history she apparently had a right eye infection following birth with atrophy. She had la grippe in 1904 and also in 1905, and influenza in 1916. She had always been subject to colds, hoarseness, bronchitis, and flatulence. She had never expectorated any milky colored fluid. No operations.

Examination showed a pale and emaciated woman past middle life appearing chronically ill, with right eye atrophied, old fracture deformity of left clavicle, slight prominence of veins of the left arm and forearm, temperature 100° F., and weighing ninety-six pounds. No enlargement of lymph glands was found. The heart was negative and blood pressure was normal. Chest examination showed as follows: left lung negative, right lung—note flat, breath sounds and vocal resonance absent below third rib and seventh vertebral spine. No râles. The abdomen was essentially negative except for flatulence. Impression on physical examination was pleurisy with effusion on right.

X-ray examination of chest on the date of admission showed old fracture deformity of left clavicle at junction of inner and middle thirds. The heart and aorta were slightly pushed to the left. Left lung: root was moderately exaggerated. There was a moderate amount of peribronchial infiltration throughout and more marked along basal trunkal markings. The parenchyma was otherwise clear. Right lung: there was an area of marked and homogeneous increase in density below the third rib and the seventh vertebral spine. There was a small amount of peribronchial infiltration above this area. The parenchyma was otherwise clear. Impression was pleurisy with effusion on right (Fig. 1).

Below follows the clinic record in brief:

Oct. 17, 1935, patient was admitted and weighed 96 pounds.

Oct. 24, right chest was aspirated and 64 ounces of milky fluid were obtained. No odor detected. Specimen was sent to laboratory.

Oct. 31, 48 ounces were aspirated and specimen was sent to laboratory. Laboratory examination of fluid gave specific gravity 1.017. Cell count was 2,350 with 8 per cent polymorphonuclears, 87 per cent small lymphocytes, 3 per cent monocytes, and 2 per cent eosinophiles. No organisms were found by Gram's or acid-fast stain. Culture of the fluid was made, but no growth was obtained. Fat by Babcock's method for butterfat was 0.9 per cent. Albumin by Purdy's method was 1.25 per cent. On centrifugation, cells did not separate from the main body of the fluid. On standing, a creamlike layer appeared at the top.

Nov. 1 and 12, sputum negative for acid-fast bacilli.

Nov. 5, 40 ounces were aspirated and specimen was sent to the laboratory. Laboratory examination showed the fat globules to stain with Sudan III. Cholesterin and lecithin crystals were not found. Total solids calculated as for cow's milk was 5.33. Protein content was 1.25 per cent.

Nov. 7, 40 ounces were aspirated. X-ray examination after removal of the fluid showed fluid level above diaphragm and a small pocket of air above. No definite pathology was visualized in the middle and lower lobes after removal of the fluid.

Nov. 8, Wassermann and Kahn tests negative. Agglutination test for Brucella melitensis and abortus negative.

Nov. 12, 26 ounces were aspirated. Urinalysis showed clear, yellow, acid, specific gravity 1.012, slight trace of albumin, sugar negative, few epithelial cells, 4 to 5 pus cells per high power field, and occasional red blood cell. Red cell count was 3,810,000. White cell count was 10,500. Hemoglobin was 70 per cent. Differential count gave

72 per cent polymorphonuclears (4 per cent stab), 19 per cent small lymphocytes, 1 per cent large lymphocytes, 3 per cent monocytes, 3 per cent eosinophiles, and 2 per cent basophiles. Oblique x-ray examination with right chest to film showed fluid level in right pleural cavity with a small pocket of pneumothorax above. No abnormal mediastinal shadows visualized. This view revealed old fracture deformities of second and fourth ribs on left.

Fig. 1.

Fig. 2. Fig. 3.

Fig. 1.—X-ray film of chest upon admittance. Effusion on right and deformity of left clavicle.

Fig. 2.—X-ray of left subclavian vein and clavicle showing deformity of clavicle, small subclavian depressed, and middle tributary vein partly filled by back pressure.

Fig. 3.—X-ray film of right subclavian vein showing wide and regular outlines though less definite.

Nov. 19, 32 ounces were aspirated.

Nov. 26, 26 ounces were aspirated.

Dec. 3, 22 ounces were aspirated. Left clavicle and subclavian vein were x-rayed following injection of one ampule of thorotrast into left cubital vein (Fig. 2).

Dec. 10, 16 ounces were aspirated. Patient complained of acute pleuritic pain in left chest.

Dec. 12, one ampule of thorotrast was injected into right cubital vein and x-ray made of right subclavian vein (Fig. 3).

Dec. 17, 20 ounces were aspirated.

Dec. 24, 10 ounces were aspirated.

Dec. 31, 2 c.c. were aspirated.

Jan. 7, 1936, no fluid obtained by aspiration. Weight was 104 pounds.

Feb. 4, examination of chest negative except for moderate impairment of note in right base and few inspiratory râles. No evidence of fluid.

April 16, x-ray examination of chest showed heart and aorta normal. Left lung: the root region was moderately exaggerated. There was moderate peribronchial infiltration extending from root region to all parts of lung and more marked along basal trunks. Right lung: the root region was moderately exaggerated, and there was a small amount of peribronchial infiltration in upper lung field and a moderate amount in lower lung field. The diaphragm was flattened. There was thickening of the pleura in the costophrenic angle.

July 2, patient stated she felt better than at any time since the accident eight years ago. Weight was 101½ pounds. Chest examination was clear except for slight impairment of percussion note below fourth rib on right. No râles. Also no difference was noted between veins of right and left arms.

DISCUSSION

Three hundred and forty-four ounces of chylous fluid were aspirated at twelve sittings over a period of sixty-eight days. There was no change in the degree of opalescence with each tapping. The amount of the fluid decreased with each successive tapping.

According to the history it appears that there was definite injury to the left subclavian vein eight years ago, and according to Lyter this is probably the most important contribution to the diagnosis of a true chylothorax. Also this case in most respects satisfies all the distinctive features of a true chylothorax when judged by the table of differential diagnostic points as given by Miller and Mohr. In addition to the history of trauma to the left subclavian vein this patient upon admission had evidence of a slight obstruction to the subclavian through definite prominence of veins of the left arm over those of the right. X-ray examination apparently confirmed this; since the picture of the left subclavian vein following intravenous dye showed a narrowing of the shadow of the vein, apparently some depression of the subclavian below the deformity of the clavicle, and narrowing of the shadows of the three veins emptying into the subclavian with a tortuosity of the superior. The middle tributary vein is filled with dye only a short distance from the subclavian, and evidently this is due to back pressure. Another x-ray plate of the left subclavian about five minutes following injection of the dye showed no residual dye. A comparison of the right subclavian vein

with that of the left shows the right to have a much wider shadow, less distinct outlines, and no depression of its shadow. The less distinct outline is probably due to lesser concentration of dye due to larger lumen of vein and faster rate of flow. (See Figs. 1 and 2.)

It is only conjecture as to when the chylothorax first appeared, but since onset of the present symptoms was January, 1935, it seems reasonable to believe that onset may have been near that time. Of course, leakage must have been slow. If this is true, there was a latent period of six and one-half years between injury and appearance of chylothorax. Strauss recently in discussing the subject with particular reference to bullet wounds states that a latent period of three days or longer elapses between trauma and appearance of chylothorax. The above case would not necessarily follow the rule, since the chylothorax in this case is probably indirectly due to trauma and may be due to scar tissue contracture around the subclavian vein or thoracic duct.

TREATMENT

The treatment of chylothorax ordinarily is unsatisfactory depending upon the cause. Depression of the clavicle causing obstruction to subclavian vein or scar tissue causing constriction of subclavian vein and thoracic duct should be more accessible and therefore more amenable to surgery. In this case it was not necessary to resort to surgery other than aspiration of the fluid. The chylous fluid gradually decreased in amount with each successive tapping, and the patient increased in weight and strength.

Appreciation is expressed to Miss Mary Foulk, clinical nurse, and Miss Katherine Morrow, technician, for their assistance.

REFERENCES

1. Lyter, J. Curtis: Chylous Ascites and Chylothorax, M. Clin. North America **11**: 479, 1927.
2. Miller, Sydney R., and Mohr, Charles F.: Diseases of Pleura, Cyclopedia of Medicine **9**: 956.
3. Strauss, Abraham: Chylothorax Due to Bullet Wounds of Thoracic Duct and Syndrome of Traumatic Chylothorax, J. THORACIC SURG. **5**: 539, 1936.
4. Cohn, Isidore: Chylothorax and Chylous Ascites, Am. J. Surg. **11**: 260, 1931.

Department of Reviews and Abstracts

Conducted by Duff S. Allen, M.D., Associate Editor

Selected Abstracts

Pleural Empyema in Children. Goran Gizelius. Acta Chir. Scandinav. **77**: 145, 1936.

The author collected 159 cases of empyema in children. In 151 cases, unilateral rib resection and drainage were instituted in 135 patients. Of these 135 patients, 115 recovered. The treatment was limited to simple aspiration in 15 patients. Five of these patients recovered, whereas the other 10 could not stand further operative intervention.

After rib resection is performed, there is no specific treatment but to watch the character of the drainage. Saline solution was used in 10 cases to irrigate the pleural cavity after operation, and vaccines were used in a few instances, but neither procedure offered better results than merely doing nothing and allowing the lung to reexpand.

The author stresses the facts that one should wait to operate until the pus is thick and the pneumonic process subsided. The patient's age was important in prognosis. The highest mortality rate occurred in infants, where 7 deaths were listed out of 14 cases. The lowest mortality rate was in the age group five to ten years old where only 7 deaths occurred in 59 cases. The bacteriology was also important in that the worst prognosis was offered in cases from which the streptococcus and mixed organisms were recovered. The commonest complication was otitis followed by nephritis and pericarditis in the order of frequency. Six cases of bilateral empyema are also reported.

Henry L. Cabitt.

Four Cases of Ewing Sarcoma in Ribs. Hilding Bergstrand. Am. J. Cancer **27**: 26, 1936.

The author reports four cases of Ewing sarcoma of the ribs. All the four patients were about the age of puberty. The tumor was localized to the sixth to eighth ribs, always posteriorly. The tumor grew into the pleural cavity, pushing the pleura away. The tumor tissue elevated the periosteum on the pleural surface of the ribs so that the

inside of the ribs was roughened with periosteal spicules, whereas the outside of the ribs was smooth although the tumor tissue had infiltrated all around the bone. The localization of the tumor to the posterior portion of the sixth to the eighth ribs is significant, since this posterior location is the site of earliest ossification. It is thus suggested that the Ewing sarcoma is due to a disturbance in the formation of the skeleton at a very early stage of fetal life.

Each of the four cases was treated by surgery, for either biopsy or removal of the tumor mass. The tumors were treated with x-radiation either before or after operative intervention. They are radiosensitive, but recurrence often follows. One of these patients treated with x-radiation and surgical extirpation is alive and well five and one-half years after operation, although metastases occurred in the lungs two years after operation and these metastases have disappeared with further x-radiation.

Henry L. Cabitt.

Readjustments in Thoracic Cage and Its Contents Following Total and Partial Pneumonectomy. W. F. Rienhoff. Southern M. J. **29**: 445, 1936.

The compensatory changes occurring in thoracic cage and contents following total and partial pneumonectomy are described. The report is based on an analysis of 36 cases. In 10 of these cases total pneumonectomy had been performed, in the remainder partial pneumonectomy. Four of the total pneumonectomies were on the right side, six on the left. The total absence of remote or immediate respiratory or circulatory embarrassment following these procedures is emphasized.

The three essential factors upon which compensatory mechanism is based are listed: (1) readjustment and adaptation of thoracic cage and diaphragm on both sides; (2) the compensatory dilatation of remaining lung; (3) the production of a space occupying fenestrated labyrinth of connective tissue which tends to fill dead space not obliterated by factors 1 and 2. Roentgenogram and drawings are presented illustrating readjustment of thoracic cage, diaphragm and remaining pulmonary tissue. A detailed description of the fibrinous mass which may develop is given.

The author believes that thoracoplasty following pneumonectomy may be unnecessary, particularly in clean cases, and certainly immediate thoracoplasty is not indicated. Phrenicectomy before or during the operation of partial or total pneumonectomy is considered unnecessary and may be harmful because of interference with normal and physiologic functions. Drainage of the empty space following pneumonectomy is to be avoided because the space is immediately

filled with coagulated mass of plasma which later organizes and becomes a fibrous tissue mass. The compensatory dilatation of the remaining lung tissue following pneumonectomy is described and distinguished from emphysema. Resection of the entire lung is considered a feasible and technically safe surgical procedure.

The author feels that patients will survive total and partial pneumonectomies in a measure comparable in mortality and postoperative incapacitation to operation for malignant visceral tumors elsewhere in the body.

Brian Blades.

Book Review

CLINICAL TUBERCULOSIS. Edited by Benjamin Goldberg, M.D., F.A.C.P., F.A.P.H.A., Associate Professor of Medicine, University of Illinois College of Medicine. Honorary Professor of Medicine, National University of Mexico. Formerly Director of the City of Chicago Municipal Tuberculosis Sanitarium. In two volumes, 640 halftone and 9 full page color plates. Philadelphia, 1935, F. A. Davis Company.

In these two volumes many contributors, all specialists in their respective fields, cover the entire subject of clinical tuberculosis. One is reminded of the famous *Handbuch der Tuberculose,* edited by Dr. L. Brauer of Hamburg, which covers this subject in the same manner. The fact that Goldberg's work is in English makes it even more valuable to physicians in this country. The encyclopedic character of the book makes it an excellent reference work in tuberculosis for all types of physicians and students. Both the medical and the surgical problems are admirably presented for the student as well as for the trained physician. It is difficult for a reviewer to discuss the numerous chapters in this book.

Dr. Godias J. Drolet, a statistician of note, describes the epidemiology of tuberculosis in a most interesting and instructive manner. Of particular interest is his study of the high rate of tuberculosis in nurses and internes who are in contact with open cases. In his opinion more care should be exercised in handling open cases. He attributes the general lowered tuberculosis mortality rate to the fact that the population is becoming adapted to the new environment of city life and that those races which are more susceptible to the disease have died in previous years.

Ralph R. Mellon discusses the B. C. G. problem. He states that sufficient evidence has accumulated that may be regarded as formidable that the B. C. G. strain of tubercle bacilli cannot be regarded as fixed virus, but is susceptible, like all other organisms, of undergoing variations as a result of which its virulence may be regained.

In the discussion on surgical collapse, Pol N. Coryllos' chapter will interest the phthisiologists who are concerned with the problem of when to recommend surgical collapse in tuberculosis. He describes the various types of surgical collapse, especially apical collapse, while the late Carl Hedblom discussed complete thoracoplasty. Coryllos states that the group statistics vary in each year, but the results on the whole are convincing that collapse therapy is here to stay and only the choice of patients and the choice of time of operations are the problem.

Numerous illustrations of various methods of collapse are included in the chapters by Coryllos. Hedblom concluded that earlier surgical treatment in selected cases will materially improve the splendid results already obtained.

Other forms of collapse are described by the Doctors Matson of Portland, Oregon. They describe in detail pneumothorax, oleothorax and cauterization of adhesions.

George G. Ornstein and David Ulmar have described physical signs in great detail in tuberculosis. Heliotherapy by Edgar Mayer and several chapters by the editor, Benjamin Goldberg, are good.

The illustrations are profuse and aid considerably in the understanding of the text.

The reviewer can recommend these volumes to medical as well as surgical colleagues.

J. J. Singer.

The American Association for Thoracic Surgery

Officers

PRESIDENT, LEO ELOESSER, SAN FRANCISCO, CALIF.
VICE PRESIDENT, STUART W. HARRINGTON, ROCHESTER, MINN.
SECRETARY, RICHARD H. MEADE, JR., PHILADELPHIA, PA.
TREASURER, EDWARD D. CHURCHILL, BOSTON, MASS.
EDITOR, EVARTS A. GRAHAM, ST. LOUIS, MO.

Council

CARL EGGERS, NEW YORK — EMILE HOLMAN, SAN FRANCISCO
FRANK B. BERRY, NEW YORK — ROBERT T. MILLER, BALTIMORE
NORMAN BETHUNE, MONTREAL

List of Members of the American Association for Thoracic Surgery, 1936

Honorary Members

BALDWIN, EDWARD R.----6 Church Street, Saranac Lake, N. Y.
CARREL, ALEXIS----Rockefeller Inst. for Med. Research, New York, N. Y.
DAVIES, H. MORRISTON----Ruthin, North Wales, Great Britain
EDWARDS, TUDOR----139 Harley Street, W. 1., London, England
KNOPF, S. ADOLPHUS----16 W. 95th Street, New York, N. Y.
LERCHE, WILLIAM----Cable, Wis.
LILIENTHAL, HOWARD----52 East 82nd Street, New York, N. Y.
MATAS, RUDOLPH----2255 St. Charles Avenue, New Orleans, La.
MEYER, ALFRED----1225 Park Avenue, New York, N. Y.
ROBERTS, J. E. H.----19 Cavendish Square, W. 1., London, England
TOREK, FRANZ----91 Lloyd Road, Montclair, N. J.

Active Members

ADAMS, WILLIAM E.----University of Chicago, Chicago, Ill.
ALEXANDER, JOHN----University Hospital, Ann Arbor, Mich.
ALLEN, CLYDE----Henry Ford Hospital, Detroit, Mich.
ALLEN, DUFF S.----Washington University Medical School, St. Louis, Mo.
AMBERSON, J. B.----Bellevue Hospital, New York, N. Y.
ANDRUS, WM. DEWITT.----New York Hospital, 525 E. 68th Street, New York, N. Y.
ARCHIBALD, EDWARD W.----52 Westmount Boulevard, Montreal, Canada
BALLON, DAVID----1471 Crescent Street, Montreal, Canada
BALLON, HARRY C.----1414 Drummond Street, Montreal, Canada
BAZIN, A. T.----1414 Drummond Street, Montreal, Canada
BECK, CLAUDE S.----Lakeside Hospital, Cleveland, Ohio

BERRY, FRANK B.----768 Park Avenue, New York, N. Y.
BETHUNE, NORMAN----Sacre Coeur Hospital, Montreal, Canada
BETTMAN, RALPH B.----104 S. Michigan Avenue, Chicago, Ill.
BIGGER, I. A.----Medical College of Virginia, Richmond, Va.
BISGARD, DEWEY----1436 Medical Arts Bldg., Omaha, Neb.
BLALOCK, ALFRED----Vanderbilt University Hospital, Nashville, Tenn.
BOHRER, JOHN V.----116 East 58th Street, New York, N. Y.
BOLAND, FRANK K.----478 Peachtree Street, N. E., Atlanta, Ga.
BRUNN, HAROLD----384 Post Street, San Francisco, Calif.
BUTLER, ETHAN FLAGG----Biggs Memorial Hospital, Ithaca, N. Y.
BYERS, J. RODDICK----3166 Westmount Boulevard, Montreal, Canada
CARLSON, HERBERT----University Hospital, Minneapolis, Minn.
CARTER, B. NOLAND----Cincinnati General Hospital, Cincinnati, Ohio
CHURCHILL, EDWARD D.----Massachusetts General Hospital, Boston, Mass.
CLERF, LOUIS H.----128 South Tenth Street, Philadelphia, Pa.
COLE, DEAN B.----Professional Building, Richmond, Va.
CORYLLOS, POL N.----48 East 75th Street, New York, N. Y.
CUTLER, ELLIOTT C.----Peter Bent Brigham Hospital, Boston, Mass.
DECKER, HARRY R.----923 Westinghouse Building, Pittsburgh, Pa.
DOLLEY, FRANK S.----427 S. Arden Boulevard, Los Angeles, Calif.
EGGERS, CARL----850 Park Avenue, New York, N. Y.
ELKIN, DAN C.----618 Doctors Building, Atlanta, Ga.
ELOESSER, LEO----490 Post Street, San Francisco, Calif.
FAULKNER, WM. B.----384 Post Street, San Francisco, Calif.
FERGUSON, R. G.----Fort San, Saskatchewan, Canada
FISHER, LINCOLN----49 Carpenter Road, Mansfield, Ohio
FLICK, JOHN B.----1608 Spruce Street, Philadelphia, Pa.
FREEDLANDER, SAMUEL O.----10515 Carnegie Avenue, Cleveland, Ohio
GALE, JOSEPH W.----1300 University Avenue, Madison, Wis.
GRAHAM, EVARTS A.----Barnes Hospital, St. Louis, Mo.
GURD, FRASER B.----Medical Arts Building, Montreal, Canada
HAIGHT, CAMERON----University Hospital, Ann Arbor, Mich.
HARRINGTON, STUART W.----Mayo Clinic, Rochester, Minn.
HART, DERL----Duke Hospital, Durham, N. C.
HARVEY, SAMUEL----New Haven Hospital, New Haven, Conn.
HAYES, JOHN N.----Rockdrift, Glenwood Estates, Saranac, N. Y.
HEAD, JEROME R.----55 East Washington Street, Chicago, Ill.
HEINBECKER, PETER----Washington University Medical School, St. Louis, Mo.
HERBERT, WILLIAM P.----Flat Iron Bldg., Asheville, N. C.
HEUER, GEORGE J.----Cornell Medical Center, New York, N. Y.
HEYD, CHARLES GORDON----116 East 53rd Street, New York, N. Y.
HOLMAN, EMILE----Stanford University, San Francisco, Calif.
HUDSON, W. A.----602 David Whitney Bldg., Detroit, Mich.
JOHNS, FRANK S.----Johnston-Willis Hospital, Richmond, Va.
KERNAN, JOHN D.----103 East 78th Street, New York, N. Y.
KINSELLA, THOMAS J.----Glenn Lake Sanatorium, Oak Terrace, Minn.
LAMBERT, ADRIAN V. S.----768 Park Avenue, New York, N. Y.
LEE, WALTER ESTELL----1833 Pine Street, Philadelphia, Pa.
LOCKWOOD, A. L.----300 Bloor Street, East, Toronto, Canada
LORD, FREDERICK T.----305 Beacon Street, Boston, Mass.
MATSON, RALPH C.----1004 Stevens Building, Portland, Ore.
MEADE, RICHARD H., JR.----2116 Pine Street, Philadelphia, Pa.
MILLER, JAMES A.----133 East 64th Street, New York, N. Y.

MITCHELL, JAMES F.—1344 Nineteenth Street, Washington, D. C.
MOORE, JULIAN A.—6 Battery Park Ave., Asheville, N. C.
MOORE, RICHMOND L.—180 Fort Washington Avenue, New York, N. Y.
MYERS, J. ARTHUR—730 LaSalle Building, Minneapolis, Minn.
NEUHOF, HAROLD—4 East 95th Street, New York, N. Y.
NEWTON, HARLAN F.—319 Longwood Ave., Boston, Mass.
O'BRIEN, EDWARD J.—10 Peterboro Street, Detroit, Mich.
OCHSNER, ALTON—Tulane University, New Orleans, La.
ORNSTEIN, GEORGE—115 Central Park West, New York, N. Y.
OVERHOLT, RICHARD H.—605 Commonwealth Avenue, Boston, Mass.
PACKARD, EDWARD N.—105 Main Street, Saranac Lake, N. Y.
PETERS, LEROY S.—221 W. Central Avenue, Albuquerque, N. M.
PETERSON, EDWARD W.—525 Park Avenue, New York, N. Y.
PICKHARDT, OTTO C.—117 East 80th Street, New York, N. Y.
PRITCHARD, STUART—W. K. Kellogg Foundation, Battle Creek, Mich.
RIENHOFF, WM. F., JR.—1201 N. Calvert Street, Baltimore, Md.
RIGLER, LEO G.—University Hospital, Minneapolis, Minn.
SCRIMGER, FRANCIS A. C.—814 Medical Arts Building, Montreal, Canada
SHIPLEY, ARTHUR M.—University Hospital, Baltimore, Md.
SINGER, JACOB J.—319 University Club Bldg., St. Louis, Mo.
SMITH, DAVID T.—Duke University, Durham, N. C.
STEINKE, CARL R.—159 S. Main Street, Akron, Ohio
STETTEN, DEWITT—850 Park Avenue, New York, N. Y.
STEWART, DAVID A.—Manitoba Sanatorium, Ninette, Canada
THEARLE, WILLIAM H.—313 First National Bank Bldg., Albuquerque, N. M.
TROUT, HUGH—Jefferson Hospital, Roanoke, Va.
TRUESDALE, PHILEMON E.—151 Rock Street, Fall River, Mass.
TUCKER, GABRIEL—326 S. 19th Street, Philadelphia, Pa.
VAN ALLEN, CHESTER M.—Sir Dorabji Tata Trust, Bombay House, Fort Bombay, India
WANGENSTEEN, OWEN H.—University Hospital, Minneapolis, Minn.
WEBB, GERALD B.—402 Burns Building, Colorado Springs, Colo.
WELLES, EDWARD S.—110 Park Avenue, Saranac Lake, N. Y.
WILENSKY, ABRAHAM O.—12 East 87th Street, New York, N. Y.
WOODRUFF, WARRINER—18 Church Street, Saranac Lake, N. Y.

Associate Members

BERCK, MAURICE—23 East 74th Street, New York, N. Y.
BIRD, CLARENCE E.—Louisville City Hospital, Louisville, Ky.
BISAILLON, JAMES MARR—1004 Stevens Bldg., Portland, Oregon
BRADSHAW, HOWARD H.—Jefferson Hospital, Philadelphia, Pa.
CABITT, HENRY L.—1248 Beacon Street, Brookline, Mass.
CARR, DUANE—899 Madison Avenue, Memphis, Tenn.
CURTIS, GEORGE M.—Ohio State University College of Medicine, Columbus, Ohio
DAVIDSON, LOUIS R.—5 East 53rd Street, New York, N. Y.
DRASH, EVERETT C.—University of Virginia Hospital, Charlottesville, Va.
FISHBACK, FREDERICK C.—1801 I Street N. W., Washington, D. C.
FINNEY, GEORGE G.—2947 St. Paul Street, Baltimore, Md.
FRANK, LOUIS WALLACE—332 West Broadway, Louisville, Ky.
GIBBON, JOHN H., JR.—1608 Spruce Street, Philadelphia, Pa.
GRAY, HOWARD K.—102 Second Avenue, S. W., Rochester, Minn.
GREENOUGH, JAMES—2 Walnut Street, Oneonta, N. Y.
HANRAHAN, EDWARD M., JR.—715 Park Avenue, Baltimore, Md.

HERBEN, GEORGE F.—Twitchell Lake, Big Moose, N. Y.
HUN, HENRY—149 Washington Avenue, Albany, N. Y.
JACKSON, CHEVALIER L.—3432 North Broad Street, Philadelphia, Pa.
JANES, ERNEST C.—250 Main Street E., Hamilton, Ont.
JANES, ROBERT M.—170 St. George Street, Toronto, Ontario, Canada
JOHNSON, HOLLIS E.—904 Medical Arts Bldg., Nashville, Tenn.
JONES, JOHN C.—427 S. Arden Blvd., Los Angeles, Calif.
KING, DONALD—Massachusetts General Hospital, Boston, Mass.
LEAHY, L. J.—105 Medical Arts Bldg., 40 North Street, Buffalo, N. Y.
LEVEN, N. LOGAN—350 St. Peter Street, St. Paul, Minn.
LONGACRE, JACOB JAMES—Cincinnati General Hospital, Cincinnati, Ohio
MACDONALD, R. H.—401-2 Canada Bldg., Saskatoon, Saskatchewan, Canada
MAYER, EDGAR—470 Park Avenue, New York, N. Y.
MCPHAIL, ROSS—Lakeview, Washington
MEYER, HERBERT WILLY—170 East 78th Street, New York, N. Y.
MEGNAULT, GEORGE—452 Sherbrooke Street, Est., Montreal, Canada
MOERSCH, HERMAN—Mayo Clinic, Rochester, Minn.
MURRAY, FOSTER—89 Hanson Place, Brooklyn, N. Y.
NEHIL, LAWRENCE W.—Midland, Michigan
NIXON, J. W.—1121 Nix Professional Bldg., San Antonio, Texas
PLUNKETT, ROBERT E.—Albany Hospital, Albany, N. Y.
POLLOCK, WM. C., MAJ. M. C., U. S. A.—Fitzsimons General Hospital, Denver, Colo.
POTTER, BENJ. P.—Hudson County Tuberculosis Hospital and Sanatorium, Secaucus, N. J.
POWERS, JOHN H.—Mary Imogene Bassett Hospital, Cooperstown, N. Y.
RINGER, PAUL H.—Arcade Bldg., Asheville, N. C.
ROGERS, W. L.—490 Post Street, San Francisco, Calif.
SHIPMAN, SIDNEY—490 Post Street, San Francisco, Calif.
SIMPSON, H. MURRAY—327 Queen's Avenue, London, Ontario, Canada
SINGLETON, ALBERT O.—320 21st Street, Galveston, Texas
SKINNER, GEORGE F.—36 Coburg Street, St. John, N. B.
STRIEDER, JOHN—171 Bay State Road, Boston, Mass.
THORBURN, GRANT—105 East 53rd Street, New York, N. Y.
TOUROFF, ARTHUR S. W.—940 Park Avenue, New York, N. Y.
TUTTLE, WM. M.—305 Professional Bldg., 10 Peterboro St., Detroit, Mich.
VAN HAZEL, WILLARD—30 N. Michigan Street, Chicago, Ill.
VIDAL, J. A.—454 Sherbrooke Street E., Montreal, Canada
WEINBERG, JOSEPH A.—1012 Medical Arts Bldg., Omaha, Neb.

Senior Members

AUCHINCLOSS, HUGH—Presbyterian Hospital, Medical Center, New York, N. Y.
BRANOWER, WILLIAM—285 Central Park West, New York, N. Y.
BROWN, LAWRASON—104 Main Street, Saranac Lake, N. Y.
CROWE, SAMUEL J.—Johns Hopkins Hospital, Baltimore, Md.
DAVISON, T. C.—478 Peachtree Street, N. E., Atlanta, Ga.
DIEDERICH, VICTOR—236 Central Avenue, Hot Springs, Ark.
DOVELL, CHAUNCEY D., MAJ. M. C., U. S. A.—Army and Navy General Hospital, Hot Springs National Park, Ark.
DUNHAM, H. KENNON—27th Floor, Union Central Life Bldg., Cincinnati, Ohio
EINHORN, MAX—20 East 63rd Street, New York, N. Y.
FISCHER, HERMAN—35 East 84th Street, New York, N. Y.
GREEN, NATHAN—New Canaan, Conn.

JACKSON, CHEVALIER----3432 North Broad Street, Philadelphia, Pa.
KELLER, WM. L., COL. M. C., U. S. A.----Walter Reed Hospital, Washington, D. C.
KENYON, JAMES----125 East 72nd Street, New York, N. Y.
LEMON, WILLIS S.----510 Tenth Avenue, S. W., Rochester, Minn.
LEWALD, LEON T.----1200 Fifth Avenue, New York, N. Y.
MCSWEENEY, E. S.----102 East 35th Street, New York, N. Y.
MAES, URBAN----1671 Octavia Street, New Orleans, La.
MANGES, MORRIS----1185 Park Avenue, New York, N. Y.
MILLER, ROBERT, JR.----Powder Point, Duxbury, Mass.
MULLER, GEORGE P.----1930 Spruce Street, Philadelphia, Pa.
PLUMMER, HENRY S.----Mayo Clinic, Rochester, Minn.
POOL, EUGENE H.----107 East 60th Street, New York, N. Y.
ROBINSON, SAMUEL----22 West Micheltorena Street, Santa Barbara, Calif.
STEWART, GEORGE A.----5300 St. Alban's Way, Baltimore, Md.
WATSON, EVERETT E.----Mt. Regis Sanatorium, Salem, Va.
WHIPPLE, ALLEN O.----180 Fort Washington Avenue, New York, N. Y.
WHITTEMORE, WYMAN----199 Beacon Street, Boston, Mass.
YATES, JOHN L.----425 East Wisconsin Avenue, Milwaukee, Wis.

Deceased Members

1920 KENNETH A. J. MACKENZIE
SAMUEL J. MELTZER
LEO S. PETERSON
1921 ROBERT M. BROWN
H. H. JANEWAY
ROBERT C. PATTERSON
JOSEPH RANSOHOFF
1922 HENRY L. LYNAH
1923 WILLIAM S. HALSTED
1927 SPARRELL S. GALE
FRED W. PARHAM
CHARLES H. PECK
1928 HOWARD A. LOTHROP
1929 GEORGES DEHELLY
1931 NORMAN B. CARSON
1932 EMIL BECK
SELLING BRILL
CHARLES LOCKWOOD
WILLY MEYER
SIDNEY YANKAUER
1934 CARL A. HEDBLOM
ALEXIS VICTOR MOSCHCOWITZ
RAY MATSON
1935 WILLIAM FRANCIS HONAN
1936 EDGAR W. PHILLIPS
HOWARD L. BEYE

Past Presidents

1918 SAMUEL J. MELTZER, New York
1919 WILLY MEYER, New York
1920 WILLY MEYER, New York
1921 RUDOLPH MATAS, New Orleans
1922 SAMUEL ROBINSON, Santa Barbara
1923 HOWARD LILIENTHAL, New York
1924 CARL A. HEDBLOM, Chicago
1925 NATHAN W. GREEN, New York
1926 EDWARD E. ARCHIBALD, Montreal
1927 FRANZ TOREK, New York
1928 EVARTS A. GRAHAM, St. Louis
1929 JOHN L. YATES, Milwaukee
1930 WYMAN WHITTEMORE, Boston
1931 ETHAN FLAGG BUTLER, Elmira, N. Y.
1932 FREDERICK T. LORD, Boston
1933 GEORGE P. MULLER, Philadelphia
1934 GEORGE HEUER, New York
1935 JOHN ALEXANDER, Ann Arbor, Mich.
1936 CARL EGGERS, New York

Past Secretaries

1918-1922 NATHAN W. GREEN, New York
1922-1924 CHARLES GORDON HEYD, New York
1924-1929 ETHAN FLAGG BUTLER, Elmira, N. Y.
1929-1935 DUFF S. ALLEN, St. Louis

Meeting Dates

1918	Chicago, June 10	1928	Washington, D. C., April 30
1919	Atlantic City, N. J., June 9	1929	St. Louis, April 25
1920	New Orleans, May 1	1930	Philadelphia, May 12
1921	Boston, June 6	1931	San Francisco, July 1
1922	Washington, D. C., April 29	1932	Ann Arbor, Mich., April 18
1923	Chicago, May 28	1933	Washington, D. C., May 9, 10, 11
1924	Rochester, Minn., June 5	1934	Boston, May 31, June 1, 2
1925	Washington, D. C., May 4	1935	New York, June 3, 4, 5
1926	Montreal, P. Q., September 30	1936	Rochester, Minn., May 4, 5, 6
1927	New York, May 9		

American Association for Thoracic Surgery to Meet at Saranac Lake, N. Y.

The Twentieth Annual Meeting of the American Association for Thoracic Surgery will be held at Saranac Lake, N. Y., on May 31, June 1 and 2, 1937.